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AND
RELATED HYDROCARBONS

A Report by the Expert Panel
on
Air Quality Criteria

COMMITTEE ON THE CHALLENGES OF MODERN SOCIETY
NORTH ATLANTIC TREATY ORGANIZATION
FEBRUARY 1974

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OTHER NATO/CCMS AIR POLLUTION PUBLICATIONS

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Proceedings of the Second Meeting of the Expert Panel on Air Pollution Modeling, N.5

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Control Techniques for Nitrogen Oxide Emissions from Stationary Sources, N.20**

Proceedings of the Fourth Meeting of the Expert Panel on Air Pollution Modeling, N.30

Symposium on Low Pollution Power Systems Development, N.32

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**French version available by Spring 1974

These documents may be obtained from the NATO Committee on the Challenges of Modern Society, 1110 Bruxelles, Brussels, Belgium, or from the Air Pollution Technical Information Center, Office of Air and Water Programs, Environmental Protection Agency, Research Triangle Park, North Carolina 27711, U.S.A. (English only.)

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FOREWORD

The material presented in this document represents an effort by the Expert Panel On Air Quality Criteria, Committee on the Challenges of Modern Society, NATO, to compile a compendium of information, using the United States' Air Quality Criteria for Photochemical Oxidants and Air Quality Criteria for Hydrocarbons as basic documents, on the effects of photochemical oxidants and related hydrocarbons in the ambient air on man and his environment.

Air quality criteria are an expression of the scientific knowledge of the relationship between various concentrations of pollutants in the air and their adverse effects on man and his environment. Air quality criteria are descriptive; that is, they describe the effects that have been observed to occur when the concentration of a pollutant in the ambient air has reached or exceeded a specific level for a specific time period. In the development of criteria, many factors, including exposure time and conditions of the environment, must be considered. The criteria must also include consideration of the contributions of all such variables to the effects of air pollution on human health, agriculture, materials, visibility, and climate. Further, the individual characteristics of the receptor must be taken into account.

This document focuses on photochemical oxidants and related hydrocarbons as found in the ambient air; therefore, literature on extremely high concentrations has not been extensively cited. The occurrence, properties, and fate of atmospheric photochemical oxidants and the principles of formation and control are reviewed in the earlier chapters; these are followed by a discussion of measurement methods and chemical interactions in the atmosphere. Effects are considered in relation to materials, vegetation, animals, and man. Reviews of both toxicological and epidemiological studies are presented. It should be noted that the reference conditions for converting units of pollutant concentration are 25°C and 760 mm Hg. Mention of commercial products does not imply endorsement by this Panel or any of its members, or of CCMS/NATO.

Recent interest and activity on the part of the public, governmental, scientific, academic, and industrial sectors of our communities have stimulated research on the effects of pollution on man and his environment. The result is an ever-increasing body of knowledge that must be analyzed, interpreted, and finally expressed as information supporting or refuting suggested criteria. The assimilation of this new knowledge is not a task that can be undertaken by a small body of men--such as a committee of this nature. Rather, the responsibility for ensuring that criteria continually reflect the most significant information available rests with those same sectors of our communities that have inspired all of us to strive to improve our environment--the public, the governmental, the scientific, the academic, and the industrial sectors.

The Panel encourages and welcomes new data, comments, and constructive criticism of this document. We suggest that a procedure be established whereby the documents can be revised and updated periodically.

At its closing session, the working group recommended that other work now going on that might be of interest to readers of this document should be mentioned. The efforts of three groups, at least, are of probable interest. A working group has been formed under the Air Management Sector Group, Environment Directorate of the Organization for Economic Cooperation and Development, to write a case history concerning photochemical oxidants. They are considering a broad range of topics such as those included in these criteria documents. Their report will probably be finished in the first half of 1974. In the United States, the National Academy of Sciences, Committee on the Medical and Biological Effects of Enviornmental Pollutants, has recently formed a panel of experts to prepare a review concerning photochemical oxidants. This review will include those topics found in criteria documents but will not be completed for some time. The National Academy of Sciences is just completing, however, an extensive report on vapor phase organic pollutants that includes volatile hydrocarbons and oxidation products. This report is expected to be available late in 1974. The National Research Council of Canada, Associate Committee on Scientific Criteria for Environmental Quality, Subcommittee on Air, is also preparing a report on oxidants.

For the Expert Panel,

F. G. Hueter, Ph.D., Chairman

ACKNOWLEDGMENT

The Chairman of the Expert Panel on Air Quality Criteria wishes to acknowledge the scientists participating from pilot, copilot, and observer countries, and the many other persons who assisted in the preparation of this document.

The working group, which performed diligently throughout the course of this effort, receives the highest praise. The group members were:

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Full recognition is given to each panel member for carrying out and coordinating review and other activities related to the development of this document in his respective country, to Dr. I. J. Buurma of the National Institute of Public Health of the Netherlands for his effort in coordinating the review and providing comments from his country, to Dr. David Shearer, Dr. David Coffin, and Mr. Rudolph Boksleitner for preparation of this document, and to staff members of the Technical Publications Branch, U. S. Environmental Protection Agency, Research Triangle Park, North Carolina, for editorial work.

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F. G. Hueter, Ph. D.

Panel Chairman

TABLE OF CONTENTS

Chapter .... Page

LIST OF FIGURES xiii
LIST OF TABLES xvi
1. HYDROCARBONS AND OTHER INTERMEDIATES IN THE PRODUCTION OF OXIDANTS IN AMBIENT AIR 1- 1
  A. INTRODUCTION 1- 1
  B. HYDROCARBON CLASSES 1- 4
  C. ATMOSPHERIC REACTIONS OF OXIDANT PRECURSORS 1- 5
  D. ESTIMATION OF HYDROCARBON EMISSIONS 1-11
    1. Emission Levels of Hydrocarbons from Natural Sources 1-11
    2. Emission Levels of Hydrocarbons from Technological Sources 1-14
  E. NATURAL SOURCES OF OZONE 1-19
  F. OZONE TRANSFER 1-21
  G. PHOTOCHEMICAL OXIDANT FORMATION PROCESSES 1-25
    1. Nitrogen Dioxide Photolytic Cycle 1-25
    2. Hydrocarbon Interaction with Nitrogen Dioxide Photolysis 1-30
    3. Hydrocarbon Reactivity 1-35
    4. Nitric Oxide and Nitrogen Dioxide 1-40
  H. EXPECTED PHOTOOXIDATION PRODUCTS 1-42
    1. Commonly Recognized Products in Photochemical Air Pollution 1-43
    2. Products of Photooxidation of Hydrocarbons in Experimental Studies 1-45
  I. METEOROLOGICAL EFFECTS 1-52
    General 1-52
    2. Sunlight 1-53
    3. Temperature 1-56
  J. REACTANT CONCENTRATION STUDIES 1-57
    1. Environmental Chamber Studies 1-57
  K FUTURE RESEARCH 1-60
  L. SUMMARY 1-64
  M. REFERENCES FOR CHAPTER 1 1-66

2. ATMOSPHERIC CONCENTRATIONS

  A. HYDROCARBONS AND RELATED COMPOUNDS 2- 1
    1. Hydrocarbons 2- 2
    2. Aldehydes 2-13
    3. Aerosols 2-14
  B. PHOTOCHEMICAL OXIDANTS 2-15
    1. Introduction 2-15
    2. Concentrations of Oxidants in Urban Atmospheres 2-16
    3. Concentrations of Peroxyacetyl Nitrate (PAN) in The Urban Atmosphere 2-36
  C. METEOROLOGICAL FACTORS 2-39
    1. General Discussion 2-39
    2. Transport 2-39
    3. Monitoring Station Location 2-42

Chapter Page

D. SUMMARY....2-44
E. REFERENCES FOR CHAPTER 2....2-47

3. MEASUREMENT METHODS....3- 1
   A. INTRODUCTION....3- 1
   B. CALIBRATION TECHNIQUES....3- 3
   C. SAMPLE COLLECTION AND HANDLING....3- 5
   D. AEROSOL MEASUREMENTS....3- 6
   E. METHODS FOR ANALYSIS OF TOTAL HYDROCARBONS....3- 7
      1. Flame Ionization....3- 7
      2. Spectrophotometric Methods....3- 8
   F. METHODS FOR ANALYSIS OF SPECIFIC HYDROCARBONS....3- 9
      1. Subtractive Columns....3- 9
      2. Gas Chromatography....3-10
      3. Spectrometric Methods....3-11
      4. Methods for Olefins....3-12
   G. METHODS FOR ANALYSIS OF GASEOUS ALDEHYDES AND KETONES...3-12
      1. General....3-12
      2. Bisulfite Method....3-12
      3. Other Condensation Reagents....3-13
   H. METHODS FOR MEASUREMENT OF TOTAL OXIDANTS....3-14
      1. Continuous Methods Utilizing Potassium Iodide....3-14
      2. Other Methods - Intermittent Sampling....3-18
   I. SPECIFIC METHODS FOR OZONE MEASUREMENT....3-19
      1. Chemiluminescence....3-19
      2. Differential Galvanic Method....3-21
      3. Ultraviolet Photometry....3-22
      4. *Trans*-butene-2 Gas-Phase Titration....3-22
      5. Other Chemical Methods....3-22
   J. METHODS FOR DETERMINATION OF PEROXYACETYL NITRATE....3-23
      1. Long-path Infrared Spectroscopy....3-23
      2. Gas Chromatography....3-24
   K. SUMMARY....3-24
   L. REFERENCES FOR CHAPTER 3....3-27

4. EFFECTS ON VEGETATION....4- 1
   A. INTRODUCTION....4- 1
   B. CLASSIFICATION OF EFFECTS....4- 3
   C. SYMPTOMS OF THE EFFECTS OF PHOTOCHEMICAL AIR POLLUTANTS ON VEGETATION....4- 4
   D. FACTORS AFFECTING RESPONSE OF VEGETATION TO PHOTOCHEMICAL AIR POLLUTANTS....4- 7
      1. Genetic Factors....4- 7
      2. Environmental Factors....4- 9
      3. Other Factors....4-12
      4. Discussion....4-16
   E. PROBLEMS IN DIAGNOSIS AND ASSESSMENT OF THE ECONOMIC IMPACT OF PHOTOCHEMICAL AIR POLLUTION ON VEGETATION....4-17
   F. DOSE-INJURY RELATIONSHIPS OF PHOTOCHEMICAL OXIDANTS AND VEGETATION....4-20

Chapter Page

G. EFFECTS OF PHOTOCHEMICAL OXIDANTS AND OZONE ON MICROORGANISMS....4-42
H. EFFECTS OF HYDROCARBONS ON MICROORGANISMS....4-47
I. RELATIVE IMPORTANCE OF HYDROCARBON GASES IN CAUSING INJURY TO VEGETATION....4-49
J. EFFECTS OF ATMOSPHERIC ALDEHYDES ON VEGETATION....4-52
K. SYMPTOMS OF EFFECTS OF ETHYLENE ON VEGETATION....4-53
L. ESTIMATES OF ECONOMIC LOSS (DAMAGE) ASSOCIATED WITH ETHYLENE INJURY TO VEGETATION....4-55
M. DOSE-INJURY RELATIONSHIPS FOR VARIOUS PLANTS EXPOSED TO ETHYLENE....4-56
N. RESEARCH NEEDS....4-60
O. SUMMARY....4-63
P. REFERENCES FOR CHAPTER 4....4-67

5. EFFECTS OF OZONE ON MATERIALS....5- 1
A. INTRODUCTION....5- 1
B. MECHANISMS OF OZONE ATTACK....5- 1
C. ELASTOMERS....5- 2
D. TEXTILES....5- 9
E. TEXTILE DYES....5-12
F. PAINTS....5-17
G. STEEL CORROSION....5-21
H. FUTURE RESEARCH NEEDS....5-24
I. SUMMARY....5-25
J. REFERENCES FOR CHAPTER 5....5-27

6. TOXICOLOGIC EFFECTS....6- 1
A. INTRODUCTION....6- 1
B. PATHOLOGICAL EFFECTS....6- 2
  1. Short-Term Exposures....6- 2
  2. Effects of Longer Exposure....6-10
C. BIOCHEMISTRY....6-13
D. PHYSIOLOGIC ALTERATION....6-20
E. EXPOSURE OF HUMAN VOLUNTEERS....6-25
F. OZONE INTERACTION WITH MICROBIAL AGENTS....6-28
  1. Ozone....6-29
  2. Photochemical Smog....6-30
  3. Mechanisms of Action....6-30
  4. Discussion....6-34
G. NON-PULMONARY EFFECTS OF OZONE....6-35
H. OZONE AS A POSSIBLE MUTAGEN....6-38
I. TOLERANCE TO OZONE....6-40
J. DISCUSSION OF EFFECTS OF OZONE....6-45
K. PEROXYACETYL NITRATES....6-50
  1. Animal Data....6-50
  2. Human Data....6-51
  3. Discussion....6-52
L. HYDROCARBONS....6-52
M. REFERENCES FOR CHAPTER 6....6-55

cont

Chapter Page

7. EPIDEMIOLOGICAL APPRAISAL OF PHOTOCHEMICAL OXIDANTS........7- 1
  - A. INTRODUCTION..7- 1
  - B. ACUTE EFFECTS OF PHOTOCHEMICAL OXIDANTS...............7- 1
    - 1. Daily Mortality in Relation to Variations in Oxidant Levels..................................7- 1
    - 2. Hospital Admissions in Relation to Oxidant Levels...7-10
    - 3. Aggravation of Respiratory Diseases by Oxidant Pollution..7-13
    - 4. Impairment of Performance Associated with Oxidant Pollution..7-26
    - 5. Eye Irritation in Relation to Variations in Oxidant Levels..7-37
  - C. CHRONIC EFFECTS OF PHOTOCHEMICAL OXIDANTS...............7-46
    - 1. Mortality in Areas of High and Low Oxidant Pollution..7-47
    - 2. General Morbidity in Areas of High and Low Oxidant Pollution................................7-51
    - 3. Effects of Photochemical Oxidant Pollution on Community Satisfaction............................7-57
  - D. SUMMARY...7-62
    - 1. Review of Results from Cited Studies...............7-62
    - 2. Future Research Needs..............................7-64
    - 3. Discussion...7-66
  - E. REFERENCES FOR CHAPTER 7..............................7-67

LIST OF FIGURES

Figure		Page
1- 1	Cumulative frequency distribution of average 15-minute ozone concentrations at Chalk River, Canada, June 1 to August 13, 1965	1-24
1- 2	Atmospheric nitrigen dioxide photolytic cycle	1-26
1- 3	Diurnal variation of NO, NO_2, and O_3 concentrations in Los Angeles, July 19, 1965	1-29
1- 4	Interaction of hydrocarbons with atmospheric nitrogen dioxide photolytic cycle	1-32
1- 5	Nitrogen dioxide dissociation as a function of wavelength of light adsorbed	1-54
1- 6	Maximum oxidant concentration as a function of propylene and nitric oxide reactant concentrations during 2-hour dynamic irradiation	1-61
1- 7	Maximum oxidant concentration as a function of hydrocarbon and nitrogen oxide concentrations reacted during 1-hour dynamic irradiation of automobile exhaust mixtures	1-61
1- 8	Maximum oxidant concentration as a function of propylene and nitric oxide reactant concentrations during static irradiations	1-61
1- 9	Maximum oxidant concentration as a function of hydrocarbon and nitrogen oxide reactant concentrations during 2-hour dynamic irradiation of automobile exhaust mixtures	1-61
2- 1	Concentration ratios for non-methane hydrocarbons/methane in Los Angeles (213 hours during October and November, 1964) and Cincinnati (574 hours during September 1964), with 655 μg/m^3 (1 ppm) methane deducted to correct for estimated background biospheric concentration	2- 3
2- 2	Nonmethane hydrocarbons by flame ionization analyzer, averaged by hour of day over several months for various cities	2- 8
2- 3	Nonmethane hydrocarbons determined by flame ionization analyzer and averaged by hour of day for three Los Angeles county sites, October 1966 through February 1967	2- 9
2- 4	Monthly variation of mean hourly oxidant concentrations for three selected cities	2-23
2- 5	Monthly variations of mean daily maximum 1-hour average oxidant concentrations for three selected U. S. cities	2-24
2- 6	Diurnal variation of mean hourly average concentrations in Los Angeles, California, and St. Louis, Missouri	2-25
2- 7	Diurnal variation of mean hourly average oxidant concentrations in Philadelphia, Pennsylvania, August 6-8, 1966	2-25
2- 8	Monthly average of hourly ozone concentration and of daily ozone maxima for Delft, 1970	2-27

Figure Page

2- 9 Diurnal variation of ozone at Frankfurt/Main and at Kleiner Feldberg mountain station on July 27 and 28, 1967 2-30
2-10 Concentration of ozone at a downtown Frankfurt/Main station and at Kleiner Feldberg mountain station on sunny days 2-31
2-10a Diurnal variation of ozone at two sites in Berlin, Germany, 1966-1967 2-32
2-11 Diurnal variation of hourly ozone concentrations in Philadelphia and Denver 2-34
2-12 Ozone concentrations measured in Amsterdam, March 10 1971 2-34
2-13 Determination of NO_2, NO, O_3, aldehyde, and hydrocarbon concentrations, University City in Rome 2-35
2-14 Diurnal variation in ozone concentration in Ankara, Turkey, on selected days of 1972 2-35
2-15 Variation of mean 1-hour average oxidant and PAN concentrations, by hour of day, in downtown Los Angeles, 1965 2-37
2-16 Variation of mean 1-hour average oxidant and PAN concentrations, by hour of day, at the Air Pollution Research Center, Riverside, California, September 1966 2-38
2-17 Monthly variation of oxidant and PAN concentrations at the Air Pollution Research Center, Riverside, California, June 1966- June 1967 2-38
2-18 Diurnal variation of mean 1-hour average oxidant concentrations at selected California sites, October 1965 2-41
2-19 Diurnal variation of mean 1-hour average carbon monoxide concentrations at selected California sites, October 1965 2-41
4- 1 Concentration of ozone versus plant response from broccoli, spinach, lettuce, and cotton, for 1-hour ozone exposures 4-32
4- 2 Time versus plant response from tomato, broccoli, and petunia to 30 pphm ozone 4-32
4- 3 Ozone concentration-time scatter diagram of 5% acute plant responses from over 100 species and varieties, calculated from data taken from references, using the equation C = a + b/T and plotting C versus T. (Dotted line represents an approximation of an acute response threshold from data presented, modified by Heck and Brandt 4-33
5- 1 Effects of sulfur dioxide and oxidant concentrations on depth of corrosion of carbon steel exposed for ten years 5-23

Figure Page

7- 1 Comparison of deaths of persons, 65 years of age and over, and maximum daily temperatures, Los Angeles County, July 1 to November 30, 1955....................7- 4

7- 2 Comparison of nursing home deaths, maximum daily temperature, and "smog alert" days in Los Angeles County, July through December 1955.....................7- 6

7- 3 Comparison of maximum concentrations of oxidant and carbon monoxide, maximum temperature, and daily death rate for cardiac and respiratory causes, Los Angeles County, 1956-1958..................................7- 8

7- 4 Fourier curves fitted to data in Figure 7-3............7- 9

7- 5 Relationship between oxidant level in the hour before an athletic event and percent of team members with decreased performance..................................7-27

7- 6 Regression curves relating eye irritation and simultaneous oxidant concentrations from a number of stations in the Los Angeles area.......................7-38

7- 7 Variation of mean maximum eye irritation, as judged by a panel of "experts" with maximum oxidant concentrations, Pasadena, August-November, 1955.......................7-38

7- 8 Relationship between oxidant concentrations and selected symptoms in Los Angeles, October 29 through November 25, 1962..................................7-41

7- 9 Mean index of eye irritation versus oxidant concentration...7-44

7-10 Relationship of high smog periods to incidence of illness and injury, persons 65 years and over, August 2-November 28, 1954..................................7-52

7-11 Relationship of high smog periods to incidence of selected conditions for persons of all ages, August 2-November 28, 1954..................................7-53

7-12 Air pollution responses for selected conditions obtained from volunteers in Los Angeles and San Francisco Bay areas...7-55

LIST OF TABLES

Table Page

1- 1 Hydrocarbon Concentrations in Morning and Afternoon Ambient Air Samples in Riverside, California, Fall 1968 .... 1- 8
1- 2 Hydrocarbon Concentrations of Irradiated and Non-irradiated Synthetic Atmospheres Containing Diluted Automobile Exhaust Gases 1- 9
1- 3 Emission Factors for Light-Duty Vehicle Exhaust Hydrocarbons by Vehicle Model Year and by Altitude 1-15
1- 4 Emission Factors for Light-Duty Vehicle Exhaust Hydrocarbons by Vehicle Model Year for State of California 1-15
1- 5 Emission Factors for Hydrocarbons From Dry-Cleaning Operations 1-16
1- 6 Emission Factors for Gaseous Hydrocarbons from Surface-Coating Operations 1-16
1- 7 Emission Factors for Hydrocarbon Evaporative Losses from the Storage of Petroleum Products 1-17
1- 8 Emission Factors for Hydrocarbon Evaporative Losses from Gasoline Marketing 1-17
1- 9 Estimated Hydrocarbon Emissions in the United States, 1970 1-18
1-10 Hydrocarbon Emissions from Transportation Sources, 1940 through 1970 (10^6 MT/year) 1-19
1-11 Estimated Hydrocarbon Emissions in the United States, 1940 through 1970 (10^6 MT/year) 1-20
1-12 Ozone or Oxidant Yields from Photooxidation of Mixtures of Organic Substance with Nitrogen Oxides in Air 1-37
1-13 Peroxyacetyl Nitrate Yields from Photooxidation of Hydrocarbon-Nitrogen Oxide Mixtures in Air 1-39
1-14 Ozone Levels Generated in Photooxidation of Various Hydrocarbons with Oxides of Nitrogen 1-48
1-15 Yields of Carbonyl Compounds in Experimental Photooxidation of Hydrocarbons with Oxides of Nitrogen 1-50
1-16 Yields of Peroxyacyl Nitrates and Alkyl Nitrates Observed in Photooxidation of Various Olefins and Alkylbenzenes 1-51
2- 1 Some Hydrocarbons Identified in Ambient Air in the United States 2- 3
2- 2 Substances Identified in Ambient Air in Zurich 2- 6
2- 3 Average Atmospheric Light Hydrocarbon Concentration by Hour, Los Angeles, September through November, 1967 2-10
2- 4 Average Atmospheric Light Hydrocarbon Concentrations by Hour, The Hague, September, 1973 2-12
2- 5 Mean of Daily Maximum Hour Average Total Hydrocarbon Concentrations (as ppm methane), 17 California Cities, 1968-1969 2-13

Table Page

2- 6 Average Aldehyde Concentrations by Hour in Los Angeles, September 25 through November 15, 1961.........2-15
2- 7 Summary of Maximum Oxidant Concentrations Recorded in Selected Cities in the United States, 1964-1967.........2-17
2- 8 Cumulative Frequency Distribution of Hourly Average Oxidant Concentrations in Selected Cities in the United States, 1964-1965..2-17
2- 9 Summary of Total Oxidant Concentrations Recorded at CAMP Sites, 1964-1972..................................2-20
2-10 Highest Monthly Mean of 1-Hour Average Oxidant Concentrations Recorded in Selected U. S. Cities, 1964 and 1965...2-22
2-11 Mean Daily Maximum of Hourly Ozone Concentrations in Delft, 1970...2-26
2-12 Monthly Averages of Ozone Concentrations in Delft, 1970.2-27
2-13 Days When The Ozone Concentration in Delft Reached or Exceeded a Peak Level of 200 μg/m^3, 1969-1971...........2-28
2-14 Days in May through July 1971 when the Ozone Concentration at One or More Sites Reached or Exceeded the Hourly Average of 200 μg/m^3...........................2-29
2-14a Monthly Averages of Ozone Concentrations at Two Stations in Berlin, Germany............................2-32
2-15 Summary of Maximum Daily 1-Hour Average Ozone Concentrations for Selected U. S. Cities..................2-33
3- 1 Effect of Nitrogen Dioxide on Oxidant Determination in the Absence of Ozone.................................3-17
3- 2 Calibration Stability of Colorimetric Oxidant Monitoring Stations, Statewide Continuous Air-Monitoring Network, California, 1963 to 1965................................3-17
4- 1 Effects of Ambient Levels of Oxidants on Vegetation.....4-21
4- 2 Effects of Long Term Controlled Ozone Exposures on Plants...4-23
4- 3 Effects of Short Term Controlled Ozone Exposures on Plant Growth and Yield...................................4-27
4- 4 Effects of PAN on Vegetation...........................4-28
4- 5 Relative Phytotoxicity of Four Members of the Peroxyacyl Nitrates, Indicated by Preliminary Fumigation on Two Species of Plants..................................4-28
4- 6 Susceptibility of Plants to Mixtures of Sulfur Dioxide and Ozone...4-29
4- 7 Threshold Susceptibility of Plants to Acute Injury From Ozone...4-35
4- 8 Projected Ozone Concentrations Which Will Produce, for Short-Term Exposures, 5 Percent Injury to Economically Important Vegetation Grown Under Sensitive Conditions...4-36

Table Page

4- 9 Projected Ozone Concentrations Which Will Produce, for Short-Term Exposures, 20 Percent Injury to Economically Important Vegetation Grown Under Sensitive Conditions...4-36
4-10 Sensitivity to Ozone of Selected Plant Species..........4-37
4-11 Summary of Effects of Ozone on Bacteria.................4-48
4-12 Relative Concentrations of Several Unsaturated Hydrocarbons that Produce Biological Response Similar to That Produced by Ethylene...........................4-50
4-13 Dosage-Response Relationships of Various Plants to Ethylene..4-61
5- 1 Formulation of Highly Ozone Sensitive Rubber............5- 6
5- 2 Effect of Ozone on Rubber...............................5- 6
5- 3 Tire Sidewall Formulation...............................5- 7
5- 4 Effects of Ozone on Sidewall Formulations Containing Various Antiozonant Concentrations.....................5- 7
5- 5 Erosion Rates of Paints Exposed to Laboratory-Controlled Ozone Environments..........................5-20
5- 6 Erosion Rate of Paints Under Field Exposure............5-22
6- 1 Summary of the Effects of Higher Concentrations of Ozone on Animals...6- 3
7- 1 Average Number of Deaths Per Day Due to Cardiac and Respiratory Causes Among Residents of Los Angeles County, Age 65 and Older, as Related to Temperature and Oxidant Concentrations by Month, 1954-1955.............7- 3
7- 2 Correlation of Morning and Early Afternoon Oxidant Levels with Oxygen Consumption and Airway Resistance of 15 Patients with Chronic Respiratory Disease.........7-19
7- 3 Proportion of Variation Associated with Environmental Factors in Symptoms, Signs and Ventilatory Tests in a Diseased and in a Control Group........................7-22
7- 4 Relationship of Average Daily Percent of Simple and Adjusted Symptoms to Photochemical Oxidant Levels (868 Days, November 1961 Through May, 1964)............7-25
7- 5 Relative Increase of Headache, Eye Discomfort, Cough, and Chest Discomfort in Relation to Photochemical Oxidant Exposure...7-25
7- 6 Hockey-Stick Threshold Functions for Simple and Adjusted[a] Symptoms in Relation to Photochemical Oxidants..7-26
7- 7 Sign-Test Data for Testing the Association of Oxidant Levels with Accidents, Los Angeles, August Through October, 1963 and 1965................................7-29
7- 8 Correlation of Eye Irritation with Simultaneous Oxidant Concentrations in Order of Decreasing Eye Irritation Score, for a Number of Stations in the Los Angeles Area.7-39

Table Page

7- 9 Correlation Between Eye Irritation and Simultaneous Other Variables as Judged by a Panel of "Experts".......7-39
7-10 Effect of Filter Upon Sensory Irritation and Chemical Measurements...7-43
7-11 Pearson Product Moment Correlation Coefficients Between Eye Irritation and Environmental Factors in a Non-filtered Room..7-44
7-12 Total Lung Cancer Mortality in an American Legion Study Population, California, 1958-1962.....................7-49
7-13 Lung Cancer Deaths and Relative Risks Per 100,000 Man-Years of an American Legion Study Population, by Extent of Cigarette Smoking and Residence, California, 1958-1962..7-49
7-14 Total Chronic Respiratory Disease Mortality in an American Legion Study Population, California, 1958-1962[a]7-50
7-15 Selected Respiratory Conditions Reported by General Population Sample, California, May 1956................7-54
7-16 Percent of Survey Responses of General and Working Populations "Bothered" by Air Pollution, by Major Geographic Areas in California, May 1956...............7-58
7-17 Percent of Survey Responses of General and Working Populations, "Bothered" by Air Pollution, Who Have Considered Moving or Changing Their Jobs for This Reason, By Major Geographic Areas in California, May 1956..7-59
7-18 Reasons For Moving From Three Areas of California in Response to General Population Survey, May 1956.........7-59
7-19 Air Pollution Effects Reported in General Population Survey, By Type of Community and by Major Georgraphic Areas in California, May 1956..........................7-60
7-20 Effects of Air Pollution on Community Satisfaction, Reported in General Population Survey, by Major Geographic Area, California, May, 1956....................7-61

CHAPTER 1

HYDROCARBONS AND OTHER INTERMEDIATES IN THE PRODUCTION OF OXIDANTS IN AMBIENT AIR

A. INTRODUCTION

Photochemical oxidants are chemical entities of concern because of their detrimental effects on biological systems and on certain materials. They are the result of sunlight-initiated, chemical reactions in the atmosphere which involve hydrocarbons and oxides of nitrogen.

The products of these reactions, termed secondary pollutants, include the oxidants. This document focuses on the photochemical oxidants and considers those hydrocarbons related to photochemical reactions.

In the original sense, the term "oxidation" describes chemical reactions in which certain atoms combine with oxygen (O_2) to form compounds known as oxides. The complete combustion of carbon in air to form carbon dioxide (CO_2) is a typical example of an oxidation reaction.

Today, the term "oxidation" is more generally used to describe the loss of one or more electrons by an atom, ion, or molecule. Conversely, a gain of electrons is referred to as "reduction." Oxidation-reduction reactions cannot be separated since in any reaction, one of the reactants is oxidized while the other is reduced; for example, in the combustion of carbon in air, carbon is oxidized while oxygen is reduced.

The purpose of this chapter is to detail the relationship of primary hydrocarbon emissions to oxidants, one of the secondary products of atmospheric reactions between hydrocarbons and certain inorganic compounds, primarily nitrogen oxides. The relationship involves the interaction of certain hydrocarbons with light-absorbing species, the subsequent oxidation of the hydrocarbons, and the accompanying formation of several new products, among which are found the oxidants, ozone and peroxyacyl nitrates.

The combustion of coal and petroleum products such as natural gas, gasoline, and fuel oil, termed "fossil fuels", is an oxidation-reduction reaction that is responsible for much of the air pollution in urban atmospheres. Fossil fuels are composed principally of hydrocarbons (RH or HC)* which, upon complete combustion, produce two oxides, carbon dioxide (CO_2) and water (H_2O). These two oxides are not considered to be air pollutants, since they are relatively nontoxic and are normal constituents of the atmosphere.

It is other byproducts of combustion in exhaust gases and the products of incomplete combustion that lead to localized air pollution problems. Because combustion processes are usually less than 100 percent efficient, combustion exhaust gases contain minor amounts of the original fuel as well as minor amounts of partially oxidized fuel. Carbon monoxide (CO), aldehydes, and unsaturated hydrocarbons are examples of products of the partial oxidation of fuel. Small amou of nitrogen, a major constitutent of air, are oxidized to nitric oxide (NO) at the high temperatures characteristic of combustion processes; and sulfur compounds, also found in varying quantities in fossil fuels, are oxidized to sulfur dioxide (SO_2). Therefore, combustion processes produce emissions that contaminate the air with oxides of carbon, nitrogen, and sulfur, in addition to a large variety of hydrocarbons and partially oxidized hydrocarbon fragments.

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*A hydrocarbon is an organic compound containing carbon and hydrogen only. The hydrocarbons are classified as alicyclic, aliphatic (acyclic), and aromatic, according to the arrangement of the atoms and the chemical properties of the compounds.

When these contaminants have been released into the atmosphere, they may react chemically to produce other contaminants quite different from those originally released. Sunlight-induced oxidation processes, termed photooxidation, are especially important in some community air pollution problems. During daylight hours, for example, NO in polluted atmospheres is rapidly oxidized to nitrogen dioxide (NO_2). Similarly, the oxidation of SO_2 to sulfur trioxide (SO_3) is accelerated and olefins and alkylbenzenes are oxidized to form aldehydes and ketones. The formation of ozone (O_3) also occurs, as well as the formation of a family of compounds identified as peroxyacyl nitrates [$R(CO)OONO_2$]. Laboratory studies suggest the presence in polluted atmospheres of peroxybenzoyl nitrate, hydrogen peroxide, and alkylhydroperoxides.

Several of the pollutants that are formed during the photochemical reaction process are termed oxidants, which are defined as atmospheric substances that will oxidize certain reagents not readily oxidized by oxygen. Because they are products of photochemical reactions, these substances are often referred to as photochemical oxidants.

Photochemical oxidants that have been measured in the atmosphere are ozone, the peroxyacyl nitrates, and nitrogen dioxide. Nitrogen dioxide will be considered here only in relation to its involvement in the formation and the measurement of other oxidants. Nitrogen dioxide is considered in detail in NATO/CCMS document N. 15.

B. HYDROCARBON CLASSES

Hydrocarbons are compounds whose molecules [illegible] of atoms of hydrogen and carbon only. All compounds of carbon, except the oxides of carbon, the carbides, and the carbonates, are organic compounds. Hydrocarbons, therefore, are organic compounds, as are many other carbon compounds having additional elements such as oxygen, nitrogen, sulfur, and chlorine. The hydrocarbons considered in this document are those gas-phase hydrocarbons that may be encountered in urban atmospheres; of special interest are those hydrocarbons emitted into the atmosphere as contaminants from technological sources.

The volatility of a hydrocarbon is roughly related to its carbon number, that is, the number of carbon atoms in each molecule. Hydrocarbons having a carbon number of 1 to 4 are gaseous at ordinary temperatures, whereas those having a carbon number of 5 or more are liquids or solids in the pure state. (Liquid mixtures of hydrocarbons, such as gasoline, may include some compounds that in the pure state would be gases or solids.) It is important to recognize, however, that some hydrocarbons with carbon numbers greater than 5 can be important gaseous air pollutants. Hydrocarbons with a carbon number greater than about 12 generally do not reach troublesome atmospheric concentrations in the gas phase; however, many of these higher hydrocarbons, as particles or in association with particulate matter, are important air contaminants in another context but are not treated in this air quality criteria document.

Hydrocarbons fall into three classes, which are defined in terms of their general molecular structure: aliphatic (acyclic), alicyclic, and aromatic. Aliphatic hydrocarbons are those whose carbon atoms are arranged in branched or unbranched chains only, without rings. Aromatic hydrocarbons are all those whose atoms are arranged in benzene rings, that is, six-membered carbon rings having only one additional atom (of hydrogen or carbon) attached to each atom in the ring. Alicyclic hydrocarbons are all those having rings other than benzene rings. A subsidiary classification often used is that of saturated versus unsaturated hydrocarbons. A saturated hydrocarbon is one in which each of the carbon atoms is bonded to four other atoms. An unsaturated hydrocarbon-which must contain two or more carbon atoms-is one in which one or more carbon atoms is bonded to fewer than four other atoms. Saturated aliphatic hydrocarbons all have the empirical formula C_nH_{2n+2}, where n is the carbon number. Saturated alicyclics with one ring have the formula C_nH_{2n}; those with two rings have the formula C_nH_{2n-2}; and so on.

C. ATMOSPHERIC REACTIONS OF OXIDANT PRECURSORS

Reactions of hydrocarbons in the urban atmosphere are important because they give rise to secondary contaminants and reaction intermediates that cause nearly all the detrimental effects of hydrocarbon air pollution. The chemistry of these reactions and their products is not fully known, but existing knowledge is sufficient to explain the most important features of the observed behavior of photochemical air pollution. In a practical sense, adequate prediction of the real effects of controlling hydrocarbon emissions depends on applying both the available knowledge and the relevant scientific principles concerning these reactions.

Hydrocarbons become involved in the photochemical air pollution complex not because of their exposure to sunlight, but because of their reactions with oxygen atoms, excited-oxygen and ozone molecules, and free radicals generated by the action of sunlight on other components in the atmosphere, particularly nitrogen dioxide. Sunlight alone has no appreciable effect on hydrocarbons in the air; in the absence of such highly reactive matter, hydrocarbons would not be involved in photochemical air pollution.

The speed with which photochemical air pollution is developed can be related directly to the rates at which hydrocarbon molecules are attacked by the reactive species and is indicated by the rate of consumption, that is, the rate of decrease in concentration of hydrocarbons. Thus, in principle, if the rate of consumption of hydrocarbons can be predicted, an expected rate of development of photochemical air pollution can be deduced.

As a consequence of the different reactivities of individual hydrocarbons, it is impossible to predict accurately the rate of consumption of hydrocarbons in photochemical air pollution reactions unless the detailed composition of the hydrocarbon component of the air is known or can be estimated. Knowledge of the total concentration of hydrocarbons is insufficient, since two atmospheres having the same total hydrocarbon measurement may contain individual hydrocarbons of very different reactivity and thus exhibit very different rates of hydrocarbon consumption and photochemical air pollution development.

The total rate of hydrocarbon consumption is the sum of the rates of consumption of all the individual hydrocarbons. Since the individual hydrocarbons have different reactivities, some are consumed much more rapidly than others, and their concentrations change correspondingly faster. Thus, the composition of the hydrocarbon component of air after an extended period of exposure to sunlight is quite different from the pre-exposure composition. Stephens and Burleson (1) compared the hydrocarbon composition of an air sample from well-aged photochemical air pollution, collected in the late afternoon, in Riverside, California, with that of early morning samples in the same vicinity. They found that in the afternoon samples, the more reactive components were reduced by more than 90 percent relative to some of the less reactive compounds (1), as shown in Table 1-1. Earlier, Neligan (2) analyzed samples of synthetic atmospheres containing diluted automobile exhaust gases before and after simulated sunlight irradiation in the laboratory, with similar results (2), as shown in Table 1-2.

The rate of consumption of each of the individual hydrocarbons is the sum of the rates of the various reactions by which it is consumed. Rates of these reactions depend in principle upon the concentrations of the individual hydrocarbon and the attacking species, and can be estimated (within reasonable limits) by established methods of theoretical chemical kinetics. A detailed treatment of the prediction of gas-phase reaction rates is provided by Benson (3), who developed methods of estimating rate parameters for many types of reactions involving carbon compounds, including hydrocarbons. These methods have been tested and found surprisingly accurate in a substantial number of reactions for

Table 1-1. HYDROCARBON CONCENTRATIONS IN MORNING AND AFTERNOON AMBIENT AIR SAMPLES IN RIVERSIDE, CALIFORNIA, FALL 1968 (1)

Hydrocarbon	Concentration, ppm	
	7:30 a.m., PST Sept. 24, 1968	4:10 p.m., PST Oct. 24, 1968
Methane	2.355	2.530
Ethane	0.0636	0.0722
Propane	0.0188	0.0499
Acetylene	0.0770	0.0420
Ethylene	0.0656	0.0179
Propylene	0.0192	0.0013
Methyl acetylene	0.0026	0.0012
1,3-Butadiene	0.0036	0.0003
1-Butene	0.0026	0.0003
Isobutene	0.0052	0.0020
trans-2-Butene	0.0014	<0.0002
cis-2-Butene	0.0014	<0.0002
2-Methyl-1-butene	0.0024	0.0004
Cyclopentene (with 2-Methyl-1, 3-butadiene)	0.0044	0.0008
trans-2-Pentene	0.0024	<0.0002
2-Methyl-2-butene	0.0026	<0.0002
Isobutane	0.0080	0.0197
Butane	0.0276	0.0620
Isopentane	0.0392	0.0412
Pentane (with 3-methyl-1-butene)	0.0224	0.0214
Cyclopentane	0.0032	0.0024
2,2-Dimethyl butane (with 1-pentene)	0.0016	0.0009
2,3-Dimethyl butane	0.0038	0.0019
2-Methyl pentane (with *cis*-2-pentene)	0.0124	0.0096
3-Methyl pentane	0.0084	0.0061
Hexane	0.0090	0.0083

Table 1-2. HYDROCARBON CONCENTRATIONS OF IRRADIATED AND NONIRRADIATED SNYTHETIC ATMOSPHERS CONTAINING DILUTED AUTOMOBILE EXHAUST GASES (2)

	Diluted-nonirradiated				Diluted-irradiated			
Sample No.	1	2	3	4	1A	2A	3A	4A
Fuel, bromine No.	20	30	30	20	20	30	30	20
Exhaust to chamber dilution ratio	1-500	1-500	1-250	1-250	1-500	1-500	1-250	1-250
Components	Concentration, ppm							
Paraffins								
Propane	0.03	0.03	0.02	0.04	0.04	0.02	0.05	0.03
2-Me-Propane	0.02	0.01	0.03	0.02	0.02	0.02	0.02	0.02
n-Butane	0.14	0.13	0.28	0.24	0.15	0.12	0.22	0.19
2-Me-Butane	0.07	0.10	0.19	0.14	0.08	0.08	0.13	0.11
n-Pentane	0.03	0.04	0.07	0.04	0.03	0.03	0.05	0.04
2-Me-Pentane 2,3-Me$_2$-Butane	0.05	0.05	0.10	0.12	0.06	0.05	0.08	0.09
n-Hexane	0.02	0.02	0.02	0.05	0.02	0.02	0.02	0.02
2,4-Me$_2$-Pentane	0.01	0.02	0.05	0.02	-	0.02	0.02	0.02
2-Me-Hexane	-	0.01	0.03	0.01	-	-	0.01	0.01
3-Me$_2$-Hexane 2,3-Me$_2$-Pentane	-	0.01	0.05	0.05	-	-	0.07	0.03
2,2,4-Me$_3$-Pentane	-	0.03	0.03	0.03	-	0.03	0.05	0.02
Total	0.37	0.45	0.87	0.76	0.40	0.39	0.72	0.58
Olefins								
Propene	0.11	0.13	0.29	0.27	0.04	0.04	0.11	0.11
2-Me-1-Propene 1-Butene	0.04	0.06	0.12	0.09	0.01	0.01	0.03	0.02
trans-2-Butene	0.02	0.01	0.03	0.02	0.005	0.01	0.03	0.02
cis-2-Butene	0.005	0.01	0.01	0.01	0.01	0.005	-	-
1-Pentene	0.02	0.005	0.01	0.005	0.005	-	0.01	-
2-Me-1-Butene	0.01	0.03	0.03	0.02	-	-	0.005	0.005
cis-2-Pentene	0.01	0.02	0.02	0.01	-	-	-	-
2-Me-2-Butene	0.03	0.06	0.10	0.05	0.01	-	0.01	-
4-Me-1-Pentene	-	0.005	-	-	-	-	-	-
1-Hexene 2-Me-1-Pentene	0.005	0.02	0.02	0.01	-	-	-	-
trans-2-Hexene *cis*-3-Hexene	0.005	0.005	0.04	0.01	-	-	-	-
cis-2-Hexene	-	0.01	0.02	0.005	-	-	-	-
Cyclopentene	-	0.01	0.01	0.01	-	-	-	-
Total	0.26	0.38	0.70	0.51	0.08	0.07	0.20	0.16
Acetylenes								
Acetylene	0.45	0.62	1.32	0.93	0.43	0.55	1.04	0.79
Me-Acetylene	0.02	0.02	0.04	0.03	0.005	0.01	0.03	0.02
Total	0.47	0.64	1.36	0.96	0.44	0.56	1.07	0.81
Diolefins								
Propadiene	-	-	-	-	-	-	-	-
1,3-Butadiene	0.02	0.04	0.06	0.04	0.005	-	0.01	0.01
Total	0.02	0.04	0.06	0.04	0.01	-	0.01	0.01

Table 1-2 (Continued). HYDROCARBON CONCENTRATIONS OF IRRADIATED AND NONIRRADIATED SYNTHETIC ATMOSPHERES CONTAINING DILUTED AUTOMOBILE EXHAUST GASES (2)

	Diluted-nonirradiated				Diluted-irradiated			
Sample No.	1	2	3	4	1A	2A	3A	4A
Components	Concentration, ppm							
Aromatics								
Benzene	0.05	0.07	0.18	0.14	0.06	0.08	0.12	0.11
Toluene	0.07	0.14	0.34	0.19	0.09	0.16	0.22	0.19
Total	0.12	0.21	0.52	0.33	0.15	0.24	0.34	0.30
Unresolved compounds								
Ethane[a]								
Ethylene	0.32	0.33	0.93	0.69	0.34	0.32	0.69	0.58
2,2-Me_2-Butane								
trans-2-Pentene	0.02	0.03	0.03	0.02	0.005	0.005	0.01	0.01
4-Me-2-Pentene								
3-Me-Pentane	0.03	0.02	0.04	0.05	0.02	0.02	0.03	0.04
Total	0.37	0.38	1.00	0.76	0.37	0.35	0.73	0.63
Cyclics								
Cyclopentane	0.01	0.01	0.02	0.02	0.01	0.005	0.01	0.01
Me-Cyclopentane	-	0.02	0.04	0.03	-	0.01	0.02	-
Cyclohexane	-	0.01	-	-	-	0.02	0.01	0.01
Total	0.01	0.04	0.06	0.05	0.01	0.04	0.04	0.02
Total hydrocarbons recovered	1.62	2.14	4.57	3.41	1.46	1.65	3.11	2.51

[a] Not detectable, but may be present in small quantities.

which experimental observations were available. Utilizing these or similar techniques, it should be possible in the near future to develop accurate and scientifically well-based means of accounting for the detailed chemical behavior of hydrocarbon gases in urban atmospheres. This will include the prediction of rates of development of secondary contaminants as well as rates of consumption of the various hydrocarbon species in the air. First steps in this direction have already been indicated by Wayne and Ernest (4) with the development of a computer simulation model for the kinetics of photooxidation of propylene.

D. ESTIMATION OF HYDROCARBON EMISSIONS

1. Emission Levels of Hydrocarbons from Natural Sources

The presence of natural hydrocarbons in the atmosphere, particularly methane, only recently has been established by measurement (5). Although most of these hydrocarbons arise from biological sources, small and highly localized quantities of methane and a few other lower-molecular-weight hydrocarbons are attributable to geothermal areas, coal fields, natural gas and petroleum fields, and natural fires.

Various estimates have been made of the contributions of these natural sources to hydrocarbons in the atmosphere. Koyama has estimated the production rate of methane to be about 2.7×10^8 metric tons (MT) per year (6). Of this amount, the greatest quantity arises from the decomposition of organic matter at the earth's surface. Ehhalt (7) and Robinson and Robbins (8), however, conclude that this is a very conservative estimate, since methane generated in swampy and tropical areas was not included.

Volatile terpenes and isoprene constitute a separate class of hydrocarbons for which worldwide natural emission rates have been estimated. Rasmussen and Went (9) have estimated the rate of production of such compounds, from vegetation, to be about 4.0×10^8 MT per year. More recently, Rasmussen (10) has estimated annual terpene emissions to be 175×10^6 MT worldwide and 17.5×10^6 MT in the United States, assuming a 75 cm canopy (uppermost spreading branchy layer of forest) depth. Hazes associated with vegetation in many areas of the world have been ascribed to photochemical aerosol formation caused by the emission of these natural products.

From these estimates, it appears that the production of methane from natural sources is roughly equivalent on a weight basis to that of the higher-molecular-weight volatile compounds. There is great difference, however, in their natural background levels, which implies a related difference in the lifetimes of these materials in their global cycles.

There is substantial agreement on the natural background concentration of methane in the atmosphere based upon different methods of measurement and the locations of such measurements. Junge (11) indicates that the worldwide range is 0.8 to 1.1 mg/m^3 (1.2 to 1.5 ppm). This is seen both in optical measurements such as reported by Fink et al. (12) and Goldberg and Muller (13) and in measurements made by gas chromatography.

From samples taken in nonurban atmospheres, Stephens and Burleson (1) report methane values averaging 0.93 mg/m^3 (1.39 ppm) over a southern California mountain area during desert winds; they reported similar values for Hawaii. Ethane, ethylene, and acetylene were found at 0.003 ppm or less. Cavanagh et al. (14) found a mean methane concentration of 1.1 mg/m^3 (1.6 ppm) with butane at 0.14 mg/m^3 (0.06 ppm) at Point Barrow, Alaska, whereas Swinnerton et al. (15), during an oceanographic cruise between Washington, D. C., and Puerto Rico, reported a mean methane concentration of 0.83 mg/m^3 (1.25 ppm). Furthermore, these latter authors found that the methane content of ocean water was in equilibrium with that found in the atmosphere.

The lowest hydrocarbon concentrations measured in urban atmospheres are in the same range as the values previously reported for methane in nonurban areas. Data on urban hydrocarbon concentrations were obtained from special gas chromatographic measurements by Altshuller et al. (16,17).

Few measurements of low-molecular-weight hydrocarbons other than methane have been made in nonurban areas, and no accurate statement can be given as to ranges of concentrations for those compounds. Butane concentrations measured at Point Barrow, Alaska, by Cavanaugh et al. (14), and ethane, ethene, and acetylene concentrations measured in southern California mountain areas by Stephens and Burleson (1) were two to three orders of magnitude lower than concentrations of methane.

Higher-molecular-weight hydrocarbon and other volatile organic compound concentrations have been estimated by Rasmussen and Went (9) on the basis of sensitive gas chromatographic measurements of alpha- and betapinene. Pinene content averaged about 0.01 ppm.

Using his own methane production figures and a total instantaneous mass of 4.3×10^{15} g in the atmosphere, Koyama (6) calculated an atmospheric average life of 20 years for methane. Ehhalt (7) and Robinson and Robbins (8) consider this to be an overestimate and suggest that an average life of only a few years is more likely. In contrast with the estimated life of methane, the lifetime of higher-molecular-weight natural hydrocarbons may be on the order of days to months.

The principal implications of the reported information on the abundance of hydrocarbons from natural sources would appear to relate to atmospheric sampling. Since methane is photochemically nonreactive and the other hydrocarbons from natural sources are too low in concentration to be of concern in urban areas, the relatively high concentration of methane as compared with other hydrocarbons of photochemical significance from technological sources makes the use of analytical methods that are specific for methane of critical importance.

It appears that in nonurban air, background levels of methane are ordinarily in the range of 0.7 to 1.0 mg/m^3 (1.0 to 1.5 ppm), whereas other hydrocarbons, including various terpenes, are each present at levels less than 0.1 ppm.

2. Emission Levels of Hydrocarbons from Technological Sources

a. Emission Factors

An adequate emission inventory will define the magnitude, frequency, duration, and relative contribution of hydrocarbon emissions from technological sources. For a regional emission inventory, a detailed analysis of all sources of interest would be desirable. It is often necessary, however, to estimate emissions from sources for which detailed data are unavailable. Estimates are obtained by the use of emission factors, which are based on sampling data, material balances, and engineering appraisals of sources similar to those in question. Tables 1-3 to 1-9 present listings of emission factors developed in the United States for selected types of sources (18).

b. Emission Estimates in the United States

Total U. S. emissions of hydrocarbons and related organic compounds to the atmosphere for the year 1970 amounted to approximately 32 x 10^6 MT (19). Table 1-9 shows the distribution of this total by major source categories. Tables 1-10 and 1-11 present hydrocarbon emission estimates for various sources for selected years from 1940 to 1970 (19).

Emission estimates (20) made for Cologne, Federal Republic of Germany, a city of 800,000 inhabitants, show 27,210 MT (30,000 tons) of hydrocarbon emitted per year over an area of 518 km^2 (200 mi^2). These studies also show that 5.7 percent of these hydrocarbon emissions arise from transportation sources, 11.4 percent from domestic heating, and 82.9 percent from industrial sources. Emission estimates in Frankfurt/Main indicate that a total of 35,000 MT/yr (38,888 tons/year) of hydrocarbons is emitted from petroleum tanks, service stations, and automobile tanks.

Table 1-3. EMISSION FACTORS FOR LIGHT-DUTY VEHICLE EXHAUST HYDROCARBONS BY VEHICLE MODEL YEAR AND BY ALTITUDE

Model year	Altitude[a]	Hydrocarbon emissions[b]	
		g/km	g/mi
Pre 1968	Low	5.5	8.8
1970		2.2	3.6
1972		1.7	2.7
Pre 1968	High	6.2	10.0
1970		3.8	6.1
1972		3.0	4.9

[a] Over 1067 m (3500 ft) above sea level considered high altitude.
[b] Excludes California data.

Table 1-4. EMISSION FACTORS FOR LIGHT-DUTY VEHICLE EXHAUST HYDROCARBONS BY VEHICLE MODEL YEAR FOR STATE OF CALIFORNIA

Model year	Hydrocarbon emissions	
	g/km	g/mi
Pre 1966	5.5	8.8
1968	2.8	4.5
1970	2.2	3.6
1972	1.7	2.7

Table 1-5. EMISSION FACTORS FOR HYDROCARBONS FROM DRY-CLEANING OPERATIONS

Degree of control	Petroleum solvents		Synthetic solvents	
	kg/MT	lb/ton	kg/MT	lb/ton
Uncontrolled	152.5	305	105	210
Average control	--	--	47.5	95
Good control	--	--	17.5	35

Table 1-6. EMISSION FACTORS FOR GASEOUS HYDROCARBONS FROM SURFACE-COATING OPERATIONS

Type of coating	Emissions	
	Kg/MT	lb/ton
Paint	560	1120
Varnish and shellac	500	1000
Lacquer	770	1540
Enamel	420	840
Primer (zinc, chromate)	660	1320

Table 1-7. EMISSION FACTORS FOR HYDROCARBON EVAPORATIVE LOSSES FROM THE STORAGE OF PETROLEUM PRODUCTS

Type of tank	Units	Type of material stored: Gasoline or finished petroleum product	Type of material stored: Crude oil
Fixed roof			
Breathing loss	lb/day-1000 gal storage capacity	0.4	0.3
	kg/day-1000 liters storage capacity	0.05	0.04
Working loss	lb/1000 gal throughput	11	8
	kg/1000 liters throughput	1.32	0.96
Floating roof			
Breathing loss	lb/day-tank	140 (40 to 210)	100 (30 to 160)
	kg/day-tank	63.5	45.4
Working loss	lb/1000 gal throughput	Neg	Neg
	kg/1000 liters throughput	Neg	Neg

Table 1-8. EMISSION FACTORS FOR HYDROCARBON EVAPORATIVE LOSSES FROM GASOLINE MARKETING

Point of emission	Emissions: kg/10^3 liters	Emissions: lb/10^3 gal
Filling of service station tanks:		
Splash fill	1.44	12
Submerged fill	0.84	7
50% splash fill and 50% submerged fill	1.08	9
Filling of automobile tanks	1.44	12

Table 1-9. ESTIMATED HYDROCARBON EMISSIONS IN THE UNITED STATES, 1970

Source category	Emissions, 10^6 MT/year			Percent of total		
Transportation	17.9			55.9		
Motor vehicles		15.15			47.9	
Gasoline			15.06			47.6
Diesel			0.09			0.3
Aircraft		0.36			1.1	
Railroads		0.09			0.3	
Vessels		0.27			0.9	
Nonhighway use of motor fuels		1.81			5.7	
Fuel combustion in stationary sources	0.54			1.7		
Coal		0.18			0.6	
Fuel oil		0.09			0.3	
Natural gas		0.27			0.8	
Wood		Neg.[a]			---	
Industrial process losses	4.99			15.8		
Solid waste disposal	1.81			5.7		
Agricultural burning	2.54			8.0		
Miscellaneous	4.08			12.9		
Forest fires[b]		0.27			0.9	
Structural fires		0.09			0.3	
Coal refuse burning		0.09			0.3	
Gasoline and solvent evaporation		3.63			11.4	
Total	33.46					

[a] Negligible (less than o.05 x 10^6 tons/year).
[b] Includes prescribed burning.

Table 1-10. HYDROCARBON EMISSIONS FROM TRANSPORTATION SOURCES, 1940 THROUGH 1970
(10^6 MT/year)

Source Category	1940	1950	1960	1968	1969	1970
Gasoline Vehicles	6.3	9.4	14.6	15.8	15.3	15.1
Diesel vehicles	Neg[a]	Neg	0.1	0.2	0.2	0.1
Locomotives	Neg	0.1	0.1	0.1	0.1	0.1
Vessels	Neg	Neg	Neg	0.3	0.3	0.3
Aircraft	Neg	0.2	0.3	0.4	0.4	0.4
Other non-highway	0.5	1.0	1.3	1.6	1.7	1.8
Total	6.8	10.7	16.4	18.4	18.0	17.8

[a] Negligible (less than 0.05×10^6 tons/year).

E. NATURAL SOURCES OF OZONE

Ozone can be formed naturally in the atmosphere by electrical discharge. The electrical energy available from the atmosphere, however, is inadequate for the formation of significant ozone concentrations over a large metropolitan area. Calculations reveal that in urban atmospheres the concentrations of ozone that can be formed from power-loss energy are several orders of magnitude smaller than 20 $\mu g/m^3$ (0.01 ppm) (21). Such a natural source of ozone, therefore, does not account for the high concentrations found in urban atmospheres.

Ozone is also formed naturally by the action of solar radiation in the stratosphere at altitudes of 15,000 to 37,000 meters (50,000 to 120,000 feet). At these altitudes, ultraviolet radiation of wavelengths shorter than 2450 Å causes oxygen molecules to photolyze to

Table 1-11. ESTIMATED HYDROCARBON EMISSIONS IN THE UNITED STATES, 1940 THROUGH 1970 (10^6 MT/year)

Source category	1940	1950	1960	1968	1969	1970
Fuel combustion in stationary sources	1.3	1.2	0.9	0.9	0.8	0.5
Transportation	6.8	10.7	16.3	18.3	18.0	17.7
Solid waste disposal	0.6	0.8	1.2	1.8	1.8	1.8
Industrial process losses	3.0	4.7	3.9	4.0	4.3	5.0
Agricultural burning	1.7	1.9	2.3	2.5	2.5	2.5
Miscellaneous	4.1	3.8	4.0	4.4	4.5	4.0
Total	17.5	23.1	28.6	31.9	31.9	31.5
Total controllable[a]	13.3	19.4	24.7	27.5	27.4	27.5

[a] Miscellaneous sources not included.

atomic oxygen. The oxygen atoms then combine with oxygen molecules in the air to form ozone.

The total amount of ozone in the upper atmosphere, as measured spectrographically, has been found to vary between 2 and 4 millimeters (22). The unit "millimeter of ozone" is the thickness the ozone layer would have if it were compressed at 1 atmosphere of pressure; 2 to 4 millimeters is equivalent, therefore, to between 490 and 980 $\mu g/m^3$ (0.25 to 0.50 ppm) at uniform distribution through a constant-density atmosphere. Maximum ozone concentrations occur at altitudes of about 20 kilometers (12 miles).

The unit "centimeter of ozone per kilometer" (cm/km) also is used to express ozone concentrations determined spectrographically. Maximum ozone concentrations as high as 0.02 cm/km occur at altitudes of about 20 kilometers (12 miles) (22).

F. OZONE TRANSFER

Several explanations have been offered for the manner in which ozone is transferred from the upper atmosphere to the lower troposphere (22,23). The transfer apparently occurs in the vicinity of the jet stream and in weather frontal zones. It is also postulated that such a transfer occurs directly across the tropopause region. The concentrations transferred to ground level, however, are not believed to be high, partly because stable air systems, including temperature inversions, serve as barriers to the descent of stratospheric ozone. Furthermore, in the troposphere, ozone is destroyed by chemical, photochemical, and catalytic reactions; clouds and combustion gases also cause the rapid dissociation of ozone.

In the early 1950's, it was believed that stratospheric ozone was a significant source of surface ozone (24,25). Several investigators have reported concentrations of ozone or of oxidant up to a few-tenths of a ppm in nonurban areas (25-27).

It is now considered impossible however, to attribute high concentrations of ozone solely to natural sources with certainty, and that limitations in methodology may have contributed to the high readings (27). In agreement with those who postulate that high concentrations of stratospheric ozone do not reach ground level, certain researchers have found very little ozone at some remote sites. In general, it can be said that oxidant concentrations in remote areas range from less than 20 to about 100 $\mu g/m^3$ (0.01 to 0.05 ppm), with most of the measurements falling between 20 and 60 $\mu g/m^3$ (0.01 and 0.03 ppm). The proportion of these ozone concentrations caused by transport from the stratosphere and the proportion caused by transport from urban areas is uncertain. In any case, however, stratospheric ozone alone cannot account for the high concentrations found in urban atmospheres.

Ozone and oxidant concentrations have been measured in a variety of nonurban locations and the reported results of a number of these studies are discussed below.

A maximum concentration of 25 $\mu g/m^3$ (0.013 ppm) was reported in Greenland in July 1960 (28). In the Antarctic, the mean monthly surface ozone values from April 1957 to May 1958 ranged from 20 to 67 $\mu g/m^3$ (0.01 to 0.034 ppm) (29). Measurements obtained at a French station in the Antartic, from February 14, 1958 to January 15, 1959, indicated that daily mean concentrations during the months of September and October were almost tenfold higher than those of April and May (30). At the Amundsen-Scott station located at the geographical South Pole, ozone was measured by means of the chemiluminescence method of Regener from 1961 through 1964, and by means of the Mast method during 1963 and 1964. The monthly mean averages ranged from about 40 to 80 $\mu g/m^3$ (0.02 to 0.04 ppm) by the former method, and from 20 to 60 $\mu g/m^3$ (0.01 to 0.03 ppm) by the latter (31). In the U. S., Haagen-Smit, using the rubber-cracking method, reported concentrations of 0 to 60 $\mu g/m^3$ (up to 0.03 ppm) in the beach and desert regions of California (32).

From December 1962 to August 1964, ozone concentrations were measured using balloon-borne chemiluminescence ozone sondes (33). Of 867 soundings up to pressure levels of 500 millibars (5570 meters or 18,280 feet at standard atmospheric conditions), 14 percent had maximum ozone concentrations greater than about 100 μg/m^3 (0.05 ppm) and 0.7 percent had maximum ozone concentrations greater than about 200 μg/m^3 (0.10 ppm). The concentration exceeded 100 μg/m^3 (0.05 ppm) most frequently at stations near large cities. The concentration exceeded 200 μg/m^3 (0.10 ppm) at two stations only, near Bedford, Massachusetts, and Seattle, Washington.

When oxidants were measured at the Petawawa Forest, Chalk River, Ontario, the daily average concentration varied from slightly under 20 μg/m^3 (0.01 ppm) to slightly over 80 μg/m^3 (0.04 ppm) (34). Measurements were made over a period of years using both the rubber-cracking method and a Mast meter, and the highest concentration measured was 120 μg/m^3 (0.06 ppm) for 4 hours. **Analysis** of diurnal fluctuations revealed that oxidant values at night were usually less than 20 μg/m^3 (0.01 ppm), increasing in the morning hours to a peak around noon of 60 μg/m^3 (0.03 ppm), and then dropping again in the evening hours. From June 1, 1965, through August 13, 1965, measurements of a 15-minute duration were made 24 hours a day for a total of 6865 measurements. The mean concentration was 22 μg/m^3 (0.011 ppm), and the maximum instantaneous reading was 120 μg/m^3 (0.06 ppm). The cumulative frequency distribution of the concentrations measured in this study is shown in Figure 1-1 (34).

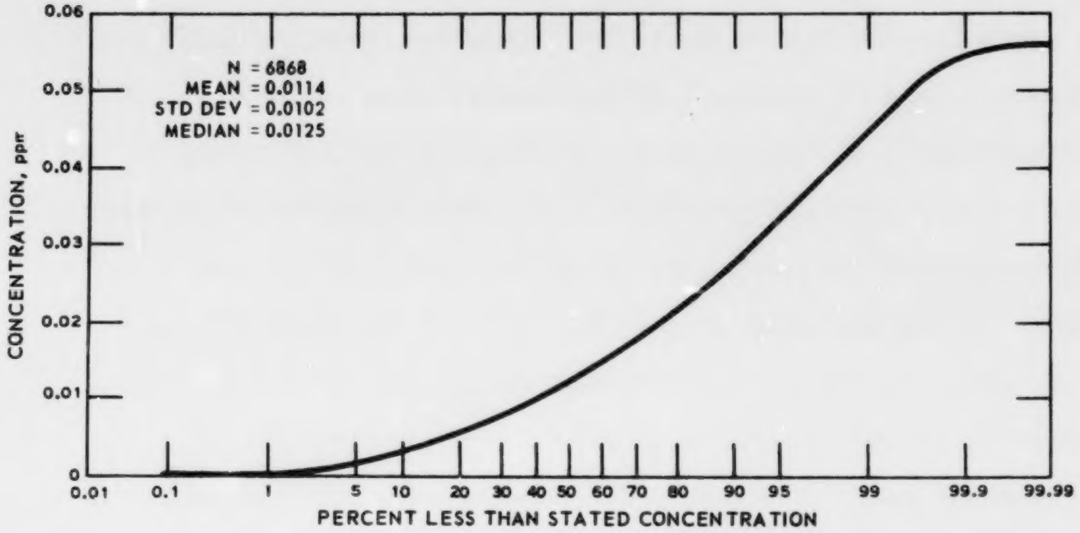

Figure 1-1. Cumulative frequency distribution of average 15-minute ozone concentrations at Chalk River, Canada, June 1 to August 13, 1965 (34).

Using Mast analyzers, Berry determined the oxidant concentrations present in valleys and on mountain tops in the southern Appalachian Mountains in the eastern United States (35). Strong diurnal variation occurred at one valley location, with the highest concentration of 80 μg/m^3 (0.04 ppm) occurring at about 1400 hr. Minimum concentrations, averaging about 29 μg/m^3 (0.015 ppm), occurred during the late evening and early morning hours. At other locations, the average oxidant concentrations generally ranged between 20 and 60 μg/m^3 (0.01 and 0.03 ppm) (35). At Green Knob, North Carolina, 2394 instantaneous readings were made every 15 minutes from June 15, 1952, through June 11, 1962. The mean concentration was 33 μg/m^3 (0.017 ppm); the maximum instantaneous reading was 140 μg/m^3 (0.07 ppm); and the standard deviation was 16 μg/m^3 (0.00790 ppm). In Pocahontas County, West Virginia, similar measurements were made from June 6 through July 6,

1961. Of data from 2880 instantaneous measurements, the mean was 49 $\mu g/m^3$ (0.025 ppm); the maximum was 125 $\mu g/m^3$ (0.064 ppm); and the standard deviation was 28 $\mu g/m^3$ (0.01475 ppm) (36).

In the summary of data by Junge (11), most of the ground-level concentrations reported were also in the range of about 20 to 60 $\mu g/m^3$ (0.01 to 0.03 ppm). Spectrographic determinations indicate that, at surface level, ozone concentrations range from 0 to 0.0004 cm/km (80 $\mu g/m^3$, or 0.04 ppm) (22).

G. PHOTOCHEMICAL OXIDANT FORMATION PROCESSES

1. Nitrogen Dioxide Photolytic Cycle

The oxidant found in the largest quantity in polluted atmospheres is ozone. In order for these observed O_3 concentrations to be generated photochemically, a process other than direct light absorption by O_2 must be involved. Light absorption by O_2 does occur in the atmosphere several miles above earth. The short ultraviolet wavelengths responsible for this reaction do not reach the earth's surface, however, and, therefore, could not produce the amounts of ozone detected in the ambient air. As indicated earlier, only a small portion of the O_3 observed near the earth's surface could have been transported from the stratosphere, where high concentrations exist naturally. Consequently, some other generation process must be involved.

Comparison of the absorption characteristics of the major atmospheric pollutants indicates that nitrogen dioxide is the most efficient absorber of the portion of the sun's ultraviolet light that reaches the earth's surface. This absorption of ultraviolet light by NO_2 leads to a complex series of reactions.

Nitrogen dioxide is broken down (photolyzed) by ultraviolet light energy into NO and O; ozone is formed in the subsequent reactions of the O atoms and O_2 (stable oxygen molecules in the air have two atoms); and new NO_2 and O_2 are generated by the reaction of NO and O_3. Thus the balance is maintained and the cycle is perpetuated. This cyclic set of reactions is depicted in Figure 1-2.

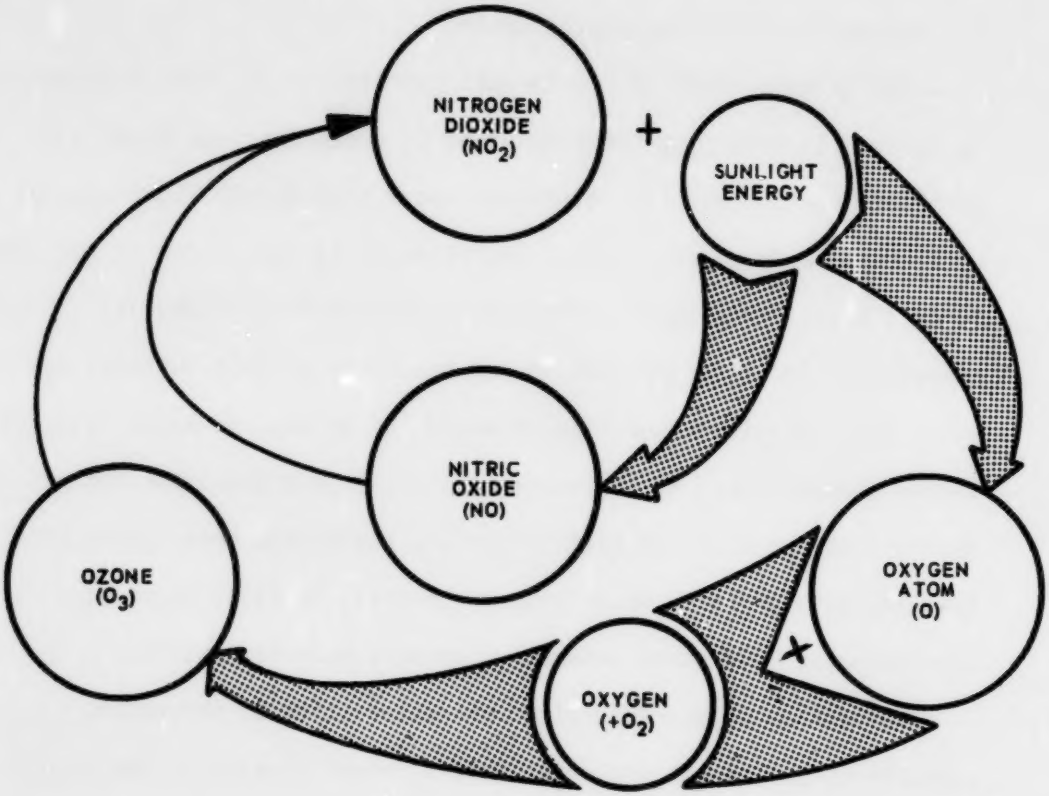
NITROGEN DIOXIDE (NO_2)
+
SUNLIGHT ENERGY
NITRIC OXIDE (NO)
OXYGEN ATOM (O)
+
OZONE (O_3)
OXYGEN ($+O_2$)

Figure 1-2. Atmospheric nitrogen dioxide photolytic cycle.

As indicated, the absorption by NO_2 of the ultraviolet portions of sunlight (3000 to 4000 Å) is followed by a set of three reactions:

$$NO_2 \xrightarrow[\text{light}]{\text{UV}} NO + O \qquad 1)$$

$$O + O_2 + M \longrightarrow O_3 + M \qquad 2)$$

$$O_3 + NO \longrightarrow NO_2 + O_2 \qquad 3)$$

These reactions may be explained as follows. In reaction 1, the energy contained in the ultraviolet light acting upon the NO_2 molecule is sufficient to break its bond, yielding two chemical substances, NO and O. The probable fate of the oxygen atom thus formed is reaction with O_2 in the atmosphere to form O_3.

Reaction 2 actually proceeds in two steps. An O atom reacts with O_2 to form an energy-rich O_3 molecule. In the absence of a means for removing this excess energy, the O_3 decomposes rapidly back to O_2 and an O atom. Collision with another molecule in the system, however, will result in the transfer of some of this excess energy, leading to the formation of a stable O_3 molecule. This collision-produced molecule is indicated by the symbol M in reaction 2. It can be any molecule in the system. In air, the collision will probably occur with N_2 or O_2, since these account for most of the molecules present.

Reaction 3 shows that the O_3 reacts with NO to reform NO_2 and O_2. Because reaction 1 is very efficient, in the absence of reactions 2 and 3, the half-life of NO_2 in the atmosphere during periods of intense sunlight would be on the order of a few minutes. Reactions 2 and 3, which result in the reformation of NO_2 in air, are also very fast, however, and tend to maintain a constant level of NO_2. Reactions 1 to 3 can be visualized as a system (Figure 1-2) in which the ultraviolet energy is acting as a pump in the rapid destruction and reformation of NO_2.

Analysis of reactions 1, 2, and 3 indicates that ozone concentrations are constrained by the steady state relationship:

$$(O_3) = k_1 I [(NO_2)/(NO)] \qquad 4)$$

In this equation, I represents the intensity of light, and k_1 is a constant whose value is determined by the absorption characteristics of NO_2 and by the rate constant for reaction 3. Parentheses are used to designate concentrations.

In the analysis of reactions 1 to 3, Leighton concludes that equation 4 must play a dominant role in determining ambient O_3 concentrations (26). This conclusion is based on the knowledge that reactions 1 to 3 are proceeding at a rate which is one to two orders of magnitude greater than any other reactions known to be occurring in the atmosphere. Thus, reactions 1 to 3 must play a dominant role in any scheme which attempts to account for ambient O_3 concentrations. The upper limit on the numerical value of the k_1I term in equation 4 is fixed by the maximum value of I. In the atmosphere, measurements show that k_1I can be no greater than 13 to 26 $\mu g/m^3$ (0.01 to 0.02 ppm). Therefore, to obtain O_3 concentrations of the order observed in the atmosphere, e.g., greater than 200 $\mu g/m^3$ (0.1 ppm), the ratio $(NO_2)/(NO)$ must be 10 or greater. In general, atmospheric measurements of O_3, NO_2, and NO during hours of sunlight confirm this prediction.

As indicated, nitric oxide and ozone cannot coexist for long because of their very fast interactions. Figure 1-3 shows, as an example of this, concentrations of certain pollutants measured in Los Angeles on July 19, 1965. The concentrations of ozone and nitric oxide did not simultaneously exceed 0.01 ppm except between 0700 and 1000 hr when concentrations of nitric oxide were rapidly decreasing and ozone rapidly increasing. The noncoexistence of high concentrations of both ozone and nitric oxide cannot always be demonstrated when 1-hour averages are used. It is possible, for example, that during the first half of an hour nitric oxide is present but ozone is not, while

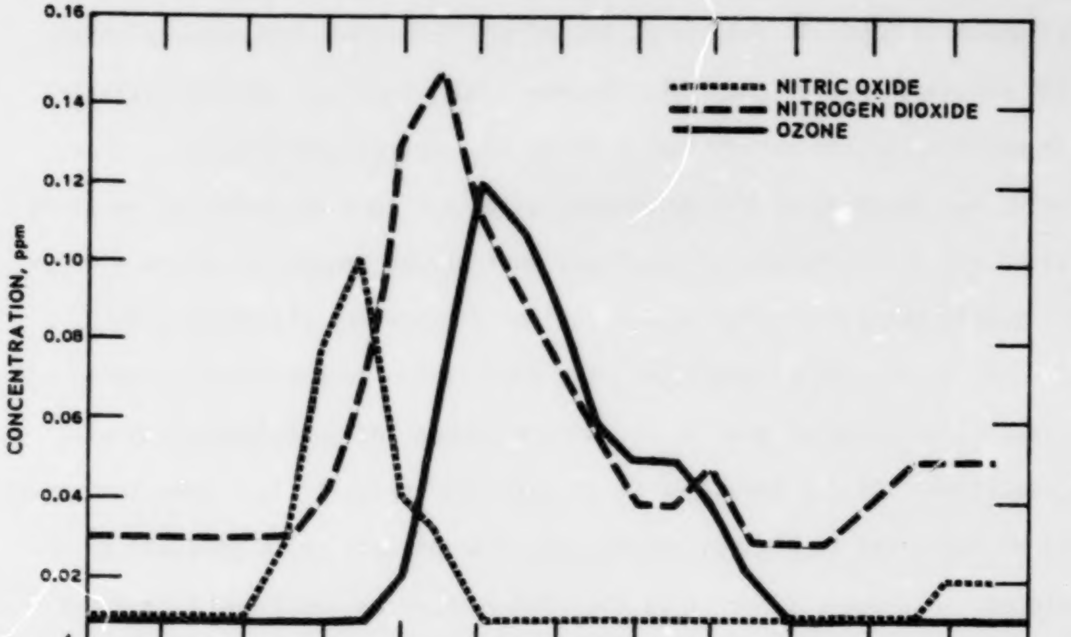

Figure 1-3. Dirunal variation of NO, NO_2, and O_3 concentrations in Los Angeles, July 19, 1965.

during the last half of the hour, ozone is present but nitric oxide is not. On the average for the hour, the presence of both will be indicated. Another factor complicating the demonstration of nonco-existence is the relatively long response time, 15 minutes or more, which is characteristic of the nitric oxide analyzer.

The changes which do occur in the atmosphere show that reactions other than 1 to 3 must also be occurring. As indicated in Figure 1-3, nitric oxide is in some instances the major oxide of nitrogen present early in the morning. Typical ratios of NO_2 to NO usually range between 0.25 and 0.50, but can reach 1 and above. As the ultraviolet light intensity increases, a rapid and almost quantitative oxidation of NO to NO_2 is observed. This photochemical oxidation is unusual since, as Leighton (26) points out, it is one of the few known instances in which the concentration of the light absorber, NO_2, increases with irradiation. In addition to the conversion of NO to NO_2, appreciable concentrations of O_3 are found after the NO concentration has been decreased as required by equation 4. Additional reactions must occur, therefore, which disrupt or modify the equilibrium concentrations dictated by reactions 1 to 3.

At steady-state conditions, the circulation scheme illustrated in Figure 1-2 indicates that O_3 and NO are formed and destroyed in equal quantities. If the amount of O_3 consumed was slightly less than the amount of NO converted to NO_2, O_3 and NO_2 would accumulate while NO would be depleted. A process slower than that represented by reactions 1 to 3 can produce such an effect if it converts NO to NO_2 without destroying an equivalent quantity of O_3.

2. Hydrocarbon Interaction with Nitrogen Dioxide Photolysis

The NO_2 photolytic cycle explains the initial formation of ozone in polluted atmospheres, but it does not explain how concentrations can develop as large as those that have been measured. If no additional mechanism were involved, most of the O_3 would quickly break down as it reacted with the NO created in NO_2 photolysis. In other words, at steady-state conditions, O_3 and NO would be formed and destroyed in equal quantities.

Laboratory experimentation and atmospheric measurements indicate that hydrocarbons provide the necessary added reactants for the atmospheric formation and buildup of NO_2 and O_3. Certain types of hydrocarbons that are also emitted into the atmosphere in fossil fuel exhausts notably olefins and substituted aromatics, enter the NO_2 circulation scheme. Studies suggest that oxygen atoms attack the hydrocarbons and the resultant oxidized compounds and free radicals react with NO to form more NO_2. Thus the balance of O_3 consumption by NO is upset so that O_3 and NO_2 levels build up while NO levels are depleted.

Figure 1-4 shows schematically how hydrocarbons are involved in photochemical oxidation reactions. Both O atoms and O_3 molecules have the ability to oxidize hydrocarbons; but, by examining rates of reactions, it has been determined that the more probable initial reaction is O atom oxidation since it is 10^8 times faster than oxidation by O_3. Although very fast, O atom oxidation is still one to two orders of magnitude slower than reaction 2. As indicated, however, processes very much slower than reaction 1 to 3 can disturb the delicate balance of the NO_2 circulation scheme illustrated in Figure 1-2.

Oxidation of hydrocarbons by O atoms is not by itself an explanation of the O_3 accumulation or of the oxidation of NO. The intermediate free radical formed from the O atom attack on hydrocarbons is quite,

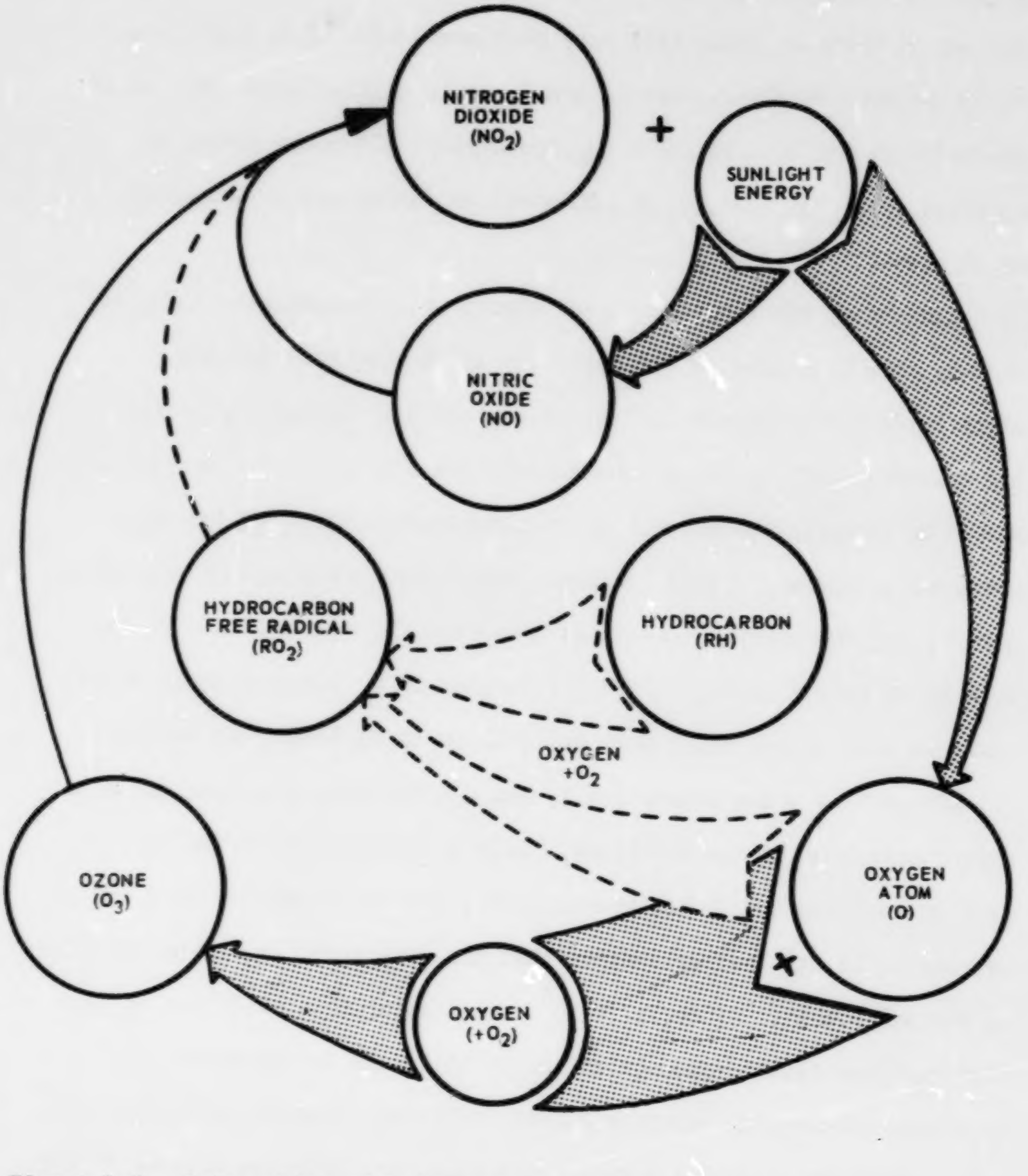
NITROGEN DIOXIDE (NO_2)
+
SUNLIGHT ENERGY
NITRIC OXIDE (NO)
HYDROCARBON FREE RADICAL (RO_2)
HYDROCARBON (RH)
OXYGEN $+O_2$
OZONE (O_3)
OXYGEN ATOM (O)
OXYGEN ($+O_2$)
X

Figure 1-4. Interaction of hydrocarbons with atmospheric nitrogen dioxide photolytic cycle.

quite reactive and apparently undergoes a series of changes in which it readily reacts with O_2 and oxidizes NO to NO_2. The effect of the hydrocarbon involvement, which adds certain postulated reactions to the NO_2 circulation scheme, is shown by the dashed lines in Figure 1-4. Examination of the indicated alternate path for the oxidation of NO to NO_2 shows that the intermediates produced by the O + RH reaction must oxidize more than one molecule of NO to NO_2. Figure 1-4 thus represents schematically the manner in which hydrocarbons enter the NO_2 circulation scheme and permit a rapid buildup of NO_2 and the accumulation of O_3.

It is important to differentiate between what is known and what is uncertain concerning the mechanism outlined in Figure 1-4. Reactions 1 to 3 have been investigated extensively and are, therefore, reasonably well understood (37). The importance of O atom and O_3 oxidation of RH also has been investigated extensively and the rate constants of these reactions are reasonably well known for a variety of hydrocarbons (26,38,39). The reactions of oxygen atoms and ozone (37,40,41), however, do not always account for the observed disappearance of hydrocarbons. Such discrepancies are probably the result of additional reactions between free-radical intermediates and hydrocarbons.

The foregoing discussion of the interaction of hydrocarbons with the NO_2 photolytic cycle should not be interpreted as invalidating the applicability of equation 4. At atmospheric O_3 concentrations below 200 $\mu g/m^3$ (0.1 ppm), however, the concurrent presence of nitric oxide and ozone occasionally can be found (42). The time-averaging period for the aerometric measurements, therefore, may be significant with respect to the reaction on a real-time basis.

In the atmosphere, the major component of the peroxyacyl nitrate series is peroxyacetyl nitrate. A small amount of peroxypropionyl nitrate has also been observed. In the laboratory, these compounds can be formed conveniently by irradiation of the parent nitrite in oxygen or air (43). This has not been proved to be a mode of formation in the atmosphere, however, since the presence of the nitrites has not been demonstrated conclusively either in the atmosphere or in laboratory systems designed to simulate photochemical air pollution systems. On the other hand, this lack of direct evidence does not eliminate nitrites as a potential source of peroxyacyl nitrates. Nitrites are rapidly photolized, and their concentrations as intermediates may not be detectable. Another possibility is that a free radical arising from an O atom attack on a hydrocarbon reacts with O_2 to form a peroxyacyl radical. The peroxyacyl radical then reacts with NO_2 to form a peroxyacyl nitrate. The peroxyacyl radical can be formed also by the reaction of O atoms with aldehydes.

It has been proposed (44) that peroxyacetyl nitrate and its homologues are formed in polluted air by a reaction between acylate (or aroylate) radicals and nitrogen trioxide. The proposed reaction accounts for the delayed formation of peroxyacetyl nitrate upon the irradiation of low concentrations of hydrocarbons and nitrogen oxides in air. It also explains why peroxyacetyl nitrate is not formed in photooxidations at high reactant concentrations. It accounts for the formation of peroxyacetyl nitrate in the dark reaction of acetaldehyde, oxygen, and N_2O_5; and, finally, it explains the inhibiting effect that excess NO_2 has on that dark reaction. The proposed reaction could yield either the accepted peroxyacetyl nitrate molecular structure or a molecular structure called acetyl pernitrate that does not include an oxygen-oxygen bond. The chemical properties of the molecule can be reconciled with either formulation.

3. Hydrocarbon Reactivity

"Hydrocarbon reactivity" is the term used to denote the relative ability of a specific hydrocarbon to exert particular effects on the photochemical reaction process. For instance, a specific hydrocarbon may be involved in several reactions in the photochemical process, depending on its concentration, structure, and oxidation state. The end products of these reactions and the consequent intensity of the symptoms generated, such as eye irritation or plant damage, are largely dependent on the nature of the hydrocarbon involved. Any "rating of reactivity" for specific hydrocarbons will, of necessity, show marked differences, depending on the effect being measured. For example, a rating based on the ability to react with O atoms will be quite different from a rating based on the capacity to generate eye irritation.

A number of schemes have been proposed for rating the reactivity of hydrocarbons (45-49). Some of these have been based on the rate of oxidation of nitric oxide to nitrogen dioxide, when the nitric oxide is irradiated with a specific hydrocarbon; the rate of disappearance of the hydrocarbon; the yield of products or effects; and, most simply, whether a hydrocarbon reacts or does not react. While there are differences in the rankings, depending on which set of criteria is used, there is general agreement that unsaturated hydrocarbons are of more importance in the production of photochemical air pollution symptoms than are saturated hydrocarbons.

Unsaturated hydrocarbons are also of great importance in the formation of oxidants. Table 1-12 presents a partial list of yields of ozone and oxidant from the photooxidation of various mixtures of organic substances and nitrogen oxides, as reported by several investigators.

Haagen-Smit (50) used the rubber cracking method to measure the ozone produced by mixtures of organic compounds and nitrogen dioxide irradiated during a 10-hour period. Schuck and Doyle (45) measured ozone with a long-path infrared spectrophotometer. The initial concentrations of the reactants irradiated were 1230 $\mu g/m^3$ (1 ppm) of nitric oxide and 3 ppm of a selected hydrocarbon (except for ethylene, of which 6 ppm was irradiated). Soon after the start of irradiation, ozone became detectable and continued to increase until a stable level was reached at the concentrations listed in Table 1-12. Altshuller and Cohen (51) and Altshuller et al. (53) used the colorimetric and coulometric potassium iodide methods for measuring oxidants.

Because of the differences in methods, concentrations of reactants, and physical properties of the environmental test chambers used, the results obtained by one set of authors are not quantitatively the same as those obtained by another, nor can they necessarily be extrapolated to atmospheric pollution conditions. They do have applicability, however, on a relative basis. Thus, in general, the internally double-bonded olefins, the diolefins, and highly substituted aromatic compounds produce the greatest yields of oxidants or ozone. Acetylene,

Table 1-12. OZONE OR OXIDANT YIELDS FROM PHOTOOXIDATION OF MIXTURES OF ORGANIC SUBSTANCE WITH NITROGEN OXIDES IN AIR

		Static irradiation yields			Dynamic irradiation yields
	Cracking depth, mm	ppm by volume			ppm by volume
Organic substance	Haagen-Smit (50)	Schuck and Doyle (45)	Altshuller and Cohen (51)	Heuss and Glasson (52)	Altshuller et al. (53)
1,3-Butadiene	12	0.65	---	0.48	0.72
2-Alkenes	8	0.55-0.73	---	0.44-0.60	---
1,3,5-Trimethylbenzene	7	---	1.1	0.46	0.37
Xylenes	6-7	0.18	0.65-1.0	0.26-0.39	---
1-Alkenes	5	0.58-1.00	---	0.41-0.54	0.4
Methanol, ethanol	5	---	---	---	---
Formaldehyde	4	---	---	---	1.05
Propionaldehyde	4	---	1.0	---	0.80
3-Methylheptane	3	---	---	---	---
n-Nonane	3	---	0.2	---	---
Ethylene	2	1.1	---	0.28	0.69
Hexanes, heptanes, iso-octane	1	0.2	---	0.2	0.0
Toluene	0.6	---	0.5	0.30	0.36
Acetylene	0.5	---	0.0	---	---
C_1-C_5 paraffins	<0.2	0.0-0.2	---	---	---

benzene, and paraffins having less than five carbon atoms produce the least amount of oxidant except at very high hydrocarbon-to-NO_x reactant ratios (45,48,50-53).

Peroxyacyl nitrate yields have been reported (45,52,54-56). Table 1-13 tabulates the reported peroxyacetyl nitrate (PAN) yields from the photooxidation of various mixtures of hydrocarbons and nitrogen oxides. Altshuller et al. (48,58) have been able to detect very low concentrations of peroxyacetyl nitrates in the 250- to 500-$\mu g/m^3$ (0.05 to 0.10 ppm) range from the irradiation of n-butane, isopentane, 2,4-dimethylhexane, and toluene with nitrogen oxides. These results indicate, as do the results of Heuss and Glasson (52), that paraffinic hydrocarbons, together with benzene and monoalkylbenzenes, contribute very little to PAN yields in atmospheric reactions. The production of PAN-type compounds is associated with the presence of propylene and higher-molecular-weight olefins, and with dialkyl- and trialkylbenzenes.

Heuss and Glasson have reported peroxybenzoyl nitrate as a minor product of the irradiation of toluene and other benzyl-type monoalkylbenzenes with nitric oxide (52). Styrene and other aromatic olefins produce somewhat greater yields of peroxybenzoyl nitrate when irradiated with nitric oxide. Peroxybenzoyl nitrate was found to be a very potent eye irritant, since only 75 to 150 $\mu g/m^3$ (0.01 to 0.02 ppm) is capable of causing light to severe eye irritation (52). This lacrimator does not appear to be formed from other types of hydrocarbons.

Table 1-13. PEROXYACETYL NITRATE YIELDS FROM PHOTOOXIDATION OF HYDROCARBON-NITROGEN OXIDE MIXTURES IN AIR (45,52,54,57)

Hydrocarbon	Peroxyacetyl nitrate concentration, ppm by volume		
	Stephens[a]	Heuss and Glasson[b]	Schuck and Doyle[c]
n-Butane	--	0	--
Isopentane	--	--	0
n-Hexane	--	0	--
Isooctane	--	0	0.05
Ethylene	0	0.01	--
Propylene	>0.55	0.35	0.35
1-Butene	0.55	0.05	0.05
Isobutene	0.15	--	0.45
1-Hexene	--	0.02	--
trans-2-Butene	--	0.63	0.52
cis-2-Butene	0.7	0.36	0.55
2-Methyl-2-butene	--	0.85	0.88
cis-3-Hexene	0.8	--	--
trans-3-Hexene	1.0	--	--
Tetramethylethylene	--	0.65	1.0
1,3-Butadiene	--	0.02	0.05
Benzene	--	0.01	--
Toluene	--	0.10	--
Monoalkylbenzenes of $>C_8$	--	0.00 or 0.01	--
o-Xylene	0.4	0.4	--
p-Xylene	0.4	0.4	--
m-Xylene	0.55	0.5	--
1,3,5-Trimethylbenzene	0.8	0.67	--
1,2,4,5-Tetramethylbenzene	0.7	--	--

[a] 5 ppm hydrocarbon and 5 ppm NO or NO_2 (References 54 and 57).

[b] 2 ppm hydrocarbon and 1 ppm NO (Reference 52).

[c] 3 pppm hydrocarbon and 1 ppm NO or NO_2 (Reference 45).

4. Nitric Oxide and Nitrogen Dioxide

One of the consequences of high-temperature combustion of fossil fuels in air is the formation of NO by oxidation of a portion of the N_2 of the air. As previously discussed, the major process by which atmospheric nitric oxide is oxidized to nitrogen dioxide is photochemical, involving hydrocarbon interaction. In addition, however, there are two other reaction pathways that contribute to the formation of atmospheric nitrogen dioxide.

The first of these, the reaction of ozone with nitric oxide, has already been considered briefly in the discussion of the nitrogen dioxide photolytic cycle. At or near sunset, when ozone photochemical formation ceases, a polluted atmosphere will generally contain an excess of ozone and a minimum of nitric oxide. While ozone formation stops at sunset, exhaust emissions of nitric oxide usually do not. From a rate-of-reaction viewpoint, the most probable fate of the remaining ozone is reaction with the continuing nitric oxide emissions. During the early evening hours, therefore, some fraction of an observed nitrogen dioxide concentration is, and would be expected to be, contributed by this reaction.

A second reaction, the oxidation of nitric oxide by oxygen in the air during the early phases of atmospheric exhaust gas dilution, is the cause of at least some of the observed nighttime nitrogen dioxide concentrations. The overall reaction, as exhaust is diluted in air, is described by the reaction:

$$2\ NO + O_2 \longrightarrow 2\ NO_2 \qquad 5)$$

The rate of formation of NO_2 from reaction 5 is determined by:

$$\frac{d(NO_2)}{dt} = k_2(NO)^2(O_2). \qquad 6)$$

When dilution is incomplete, that is, when the NO concentration is in the ppm range, reaction 5 will result in a small contribution to the oxidation of nitric oxide. Since the dilution of atmospheric exhaust gas occurs very rapidly, the amount of NO oxidized by this process is usually less than 10 percent of the total NO emissions from sunset to sunrise. Consequently, it is not unusual to have the maximum daily NO concentration recorded early in the morning.

5. Singlet Oxygen

According to Pitts (59), an important role is played by singlet molecular oxygen $O_2(^1\Delta_g)$, which can be formed in polluted urban atmosphere by:

1. Direct absorption by ground-state molecular oxygen ($O_2{}^3\Sigma_g$).
2. Absorption of solar radiation by organic pollutant molecules, such as aldehydes, ketones, or polynuclear aromatic hydrocarbons, followed by the transfer of energy to ground-state molecular oxygen.
3. Photodecomposition of ozone.
4. Absorption of ultraviolet light by short-lived oxygen-olefin or oxygen-aromatic hydrocarbon collision complexes.
5. Photoexcitation of NO_2 (60).

Singlet molecular oxygen may also be emitted from certain exothermic chemical reactions.

In urban atmospheres, singlet molecular oxygen may influence the the photoconversion of nitric oxide to nitrogen dioxide. Singlet molecular oxygen ($^1O_2$) formed by energy transfer from a donor organic molecule will react with olefinic hydrocarbons to give hydroperoxides; these compounds are thermally unstable, sensitive to surface catalysts, and photochemically reactive ($RCOOH + hv \longrightarrow RCO+OH$). They decompose to give free radicals of the same type as those formed in the direct attack of atomic oxygen on olefins and aldehydes. These reactions resemble those of the NO_x-olefin-air system and could occur simultaneously. The involvement of $^1O_2$ would be a concurrent process, admittedly possibly minor, to the overall reaction.

H. EXPECTED PHOTOOXIDATION PRODUCTS

The ultimate products of photooxidation of hydrocarbons in urban air, if a captured parcel of air were to be irradiated by sunlight for a long enough time, would probably be carbon dioxide and water vapor. From the standpoint of ordinary urban atmospheres, these products are of no interest because, aside from their abundance in the natural global atmosphere, no air parcel remains in the urban atmosphere long enough for its hydrocarbons to be fully oxidized. The products of concern in photochemical air pollution are all intermediate products of the complex, that is, all are capable of further reaction and degradation. They differ in that some react very slowly and therefore accumulate to appreciable concentrations only if formed at appreciable rates, whereas others react rapidly and are therefore efficiently scavenged, even though their rates of formation may be high. In the following paragraphs, the confirmed and expected intermediate and end products of the photochemical complex are discussed, beginning with those having the longest lives and those occurring in the highest concentrations.

1. Commonly Recognized Products in Photochemical Air Pollution

a. Ozone

Best known of the compounds generated in photochemical air pollution is ozone (O_3), a highly reactive and toxic form of oxygen. Ozone plays a significant role in the degradation of hydrocarbons, especially the olefinic hydrocarbons. The level and composition of the hydrocarbons in the urban atmosphere, in conjunction with levels of oxides of nitrogen and meteorological factors (including sunlight), determine the concentrations and dosages of ozone that will be reached in photochemical episodes, as they do those of all the secondary contaminants.

b. Nitrogen Dioxide

Nitrogen dioxide in photochemical smog is generated primarily by the photooxidation of nitric oxide, which is facilitated by the free radicals generated by photolysis of aldehydes and other secondary contaminants, as well as by the reactions of hydrocarbons with oxygen atoms and other chemical species.

c. Aldehydes

Aldehydes are major products in the photooxidation of hydrocarbons and in the individual reactions of hydrocarbons with ozone, oxygen atoms, or free radicals. They are dissociated by the ultraviolet fraction of sunlight with the production of alkyl radicals, formyl radicals, and hydrogen atoms. Because the chemical bond between the hydrogen atom and the carbonyl group is weaker than most carbon-hydrogen bonds in hydrocarbon molecules, aldehydes are relatively more susceptible to hydrogen abstraction reactions, which yield acyl radicals or alkyl radicals and carbon monoxide, according to the equations:

$$CH_3CHO + RO \longrightarrow CH_3CO + ROH \qquad 7)$$

$$CH_3CO + M \rightarrow CH_3 + CO + M \qquad 8)$$

In urban air, however, the photolysis and all other aldehyde-consuming reactions such as reaction 7 are not collectively rapid enough to prevent the accumulation of aldehydes to levels as high as about 0.2 ppm in photochemical air pollution (61).

Specific aldehydes found in urban air are formaldehyde ($HCHO$) and acrolein (CH_2CHCHO). Formaldehyde usually accounts for about 50 percent of the estimated total aldehydes in polluted air, and acrolein for perhaps 5 percent (62,63). The chemical identity of the remaining aldehydes in urban air is unknown, but higher aliphatic aldehydes such as acetaldehyde (CH_3CHO) are probably present.

d. Peroxyacyl Nitrates

Peroxyacyl nitrates, $R(CO)OONO_2$, are in the main probably products of the reaction of peroxyacyl radicals with nitrogen dioxide, as in equation 9,

$$R(CO)OO + NO_2 \longrightarrow R(CO)OONO_2 \qquad 9)$$

although other reactions, such as acylate radicals with nitrogen trioxide, as in equation 10, are not completely excluded.

$$R(CO)O + NO_3 \longrightarrow R(CO)OONO_2 \qquad 10)$$

These compounds are among the products of photochemical air pollution that oxidize certain standard reducing agents. It should be noted that the most abundant member of the class, peroxyacetyl nitrate (PAN), is the only one usually detected in atmospheric samples, although smaller amounts of higher homologues are probably present.

PAN may absorb ultraviolet light from the sun, with probable production of free radicals. The specific products of the primary step are not known.

2. Products of Photooxidation of Hydrocarbons in Experimental Studies

Laboratory irradiations of synthetic atmospheres provide valuable information in understanding the photochemistry of polluted air because they facilitate the study of rates and products of reactions of individual hydrocarbons or of mixtures with carefully controlled composition. Further, they can be used to determine quantitatively the relationship between reaction rates or product yields and such environmental factors as light intensity, temperature, humidity, and initial reactant concentrations. In many such studies the initial concentrations employed have been larger than those usually found in urban atmospheres, but still small enough to give a reasonably good indication of the relations to be expected under conditions prevalent in the ambient atmosphere.

The many studies of irradiated synthetic atmospheres containing hydrocarbons and oxides of nitrogen have been thoroughly reviewed by Altshuller and Bufalini (41). From that review and reports of subsequent studies (48,58), it appears that the carbon atoms of photooxidized hydrocarbons can be well accounted for in terms of aldehydes, ketones, carbon monoxide, carbon dioxide, alkyl nitrates, and peroxyacyl nitrates, except that acceptable carbon balances have not been achieved for the photooxidation of aromatic hydrocarbons.

The chemical behavior of irradiated atmospheres containing auto exhaust has been shown to resemble rather closely that of photooxidized mixtures of hydrocarbons and nitric oxide. Ozone and peroxyacyl nitrates are formed but do not accumulate until nitric oxide is nearly consumed, whereas aldehydes are produced from the beginning of the irradiation and are always found as major products.

a. Ozone

Appreciable ozone concentrations have been observed to result from photooxidation (with nitric oxide or nitrogen dioxide) of many olefins, paraffins (not including methane), and alkylbenzenes. At concentrations of 3 ppm hydrocarbons and 1230 $\mu g/m^3$ (1 ppm) nitrogen oxide, Schuck and Doyle (45) measured ozone levels between 200 and 1960 $\mu g/m^3$ (0.1 and 1 ppm) for 25 hydrocarbons, as listed in Table 1-14. At the selected initial concentrations, most of the olefins tested yielded more than 980 $\mu g/m^3$ (0.5 ppm), while most of the paraffins yielded less than that amount. At other initial concentrations, there can be little doubt that different results would be obtained. Altshuller et al. (48) showed, for example, that in experiments with 3460 $\mu g/m^3$ (2 ppm) propylene the optimum nitric oxide level for ozone production is about 1230 $\mu g/m^3$ (1 ppm), which yields 2160 $\mu g/m^3$ (1.1 ppm) ozone; while for 6870 $\mu g/m^3$ (3 ppm) butane, the optimum nitric oxide level is about 125 $\mu g/m^3$ (0.1 ppm), which yields about 1760 $\mu g/m^3$ (0.9 ppm) ozone.

A simple relation that would permit the prediction of ozone yields, either absolute or relative, from initial concentrations in irradiation experiments has not yet been discovered. There appears to be a practical limit to the concentration of ozone that can be generated in this way; regardless of what hydrocarbon or mixture of hydrocarbons serves as the substrate for photooxidation, and regardless of what initial concentrations or light intensities have been provided, no chamber irradiation has yet produced ozone in excess of 2940 $\mu g/m^3$ (1.5 ppm), and only a few have reached 1960 $\mu g/m^3$ (1.0 ppm). In urban atmospheres, a level as high as 1960 $\mu g/m^3$ (1.0 ppm) has not yet been recorded.

b. Nitrogen Dioxide

All irradiation chamber photooxidations in which nitric oxide is introduced as a reactant show conversion of nitric oxide to nitrogen dioxide at rates dependent on the reactant levels and the hydrocarbon reactivity as well as on such experimental variables as light intensity and temperature. Ordinarily, the nitrogen dioxide concentration reaches a maximum and then declines somewhat less rapidly than it had risen. The time required for the nitrogen dioxide concentration to reach this maximum is always less than the time required for ozone to reach a maximum. Some examples are seen in Table 1-14. The degree of conversion (that is, the ratio of highest observed nitrogen dioxide concentration to the initial total of both oxides) is often near 100 percent, especially in systems in which the reactions are rapid, such as systems containing the more reactive olefins and nitric oxide concentrations near the optimum for ozone production.

Table 1-14. OZONE LEVELS GENERATED IN PHOTOOXIDATION[a] OF VARIOUS HYDROCARBONS WITH OXIDES OF NITROGEN (45)

Hydrocarbon	Ozone level, ppm	Time, min
Isobutene	1.00	28
2-Methyl-1,3-butadiene	0.80	45
trans-2-Butene	0.73	35
3-Heptene	0.72	60
2-Ethyl-1-butene	0.72	80
1,3-Pentadiene	0.70	45
Propylene	0.68	75
1,3-Butadiene	0.65	45
2,3-Dimethyl-1, 3-butadiene	0.65	45
2,3-Dimethyl-2-butene	0.64	70
1-Pentene	0.62	45
1-Butene	0.58	45
cis-2-Butene	0.55	35
2,4,4-Trimethyl-2-pentene	0.55	50
1,5-Hexadiene	0.52	85
2-Methylpentane	0.50	170
1,5-Cyclooctadiene	0.48	65
Cyclohexene	0.45	35
2-Methylheptane	0.45	180
2-Methyl-2-butene	0.45	38
2,2,4-Trimethylpentane	0.26	80
3-Methylpentane	0.22	100
1,2-Butadiene	0.20	60
Cyclohexane	0.20	80
Pentane	0.18	100
Methane	0.0	

[a] Hydrocarbon concentration (initial) 3 ppm; oxide of nitrogen (NO or NO_2, initial) 1 ppm.

c. Aldehydes and Ketones

In all irradiation chamber experiments in which products have been appropriately analyzed, carbonyl compounds have been found among the major products. Table 1-15 shows yields of these compounds for a number of initial hydrocarbons, as tabulated by Altshuller and Bufalini (41). The yields are shown in relation to initial concentrations in the irradiation chamber; therefore, yields relative to hydrocarbons actually reacted would be greater than those shown (although perhaps, in some cases, not much greater).

Table 1-15 shows formaldehyde to be the most important carbonyl product resulting from the photooxidation of many of these compounds, forming half or more of the total for each of the 1-alkenes. Acetaldehyde is important as a product from propylene, cis- and trans-2-butenes, and 2-methyl-2-butene, but not from ethylene, isobutene, 1,3-pentadiene or 2-methyl-1,3 pentadiene; acetone, is an important product from isobutene, 2-methyl-2-butene, and 2,3-dimethyl-2-butene. For the olefins cited, the results are consistent with the view that a large fraction of the hydrocarbon molecules that react yield products typical of their reaction with ozone. It appears, however, that some of the formaldehyde ultimately produced may result from the further reactions of other intermediate species in the system, including higher aldehydes.

With alkylbenzenes, the yield of carbonyl compounds is smaller (54), ranging from 0.1 to 0.3 relative to the hydrocarbon reacted. Again, most of the carbonyl product is formaldehyde, with smaller amounts of acetaldehyde reported.

Paraffins also yield carbonyl compounds upon photooxidation. Using 3 ppm of hydrocarbon with 0.6 ppm of nitrogen oxides (nitrogen oxide composition not specified), Altshuller et al. (48) found formaldehyde, acetaldehyde, propionaldehyde, butanone (methyl ethyl ketone), and a component that may have been acetone, butyraldehyde, or both. Alkanes tested in this study were butane, isopentane, 2-methylpentane, 3-methylhexane, 2,4-dimethylhexane, n-nonane, and methylcyclohexane. About 70 percent of the isopentane consumed could be accounted for as carbonyl products.

Table 1-15. YIELDS OF CARBONYL COMPOUNDS IN EXPERIMENTAL PHOTOOXIDATION OF HYDROCARBONS WITH OXIDES OF NITROGEN (41)

Hydrocarbon	Moles/mole of initial hydrocarbon				Total or other aldehydes
	Formaldehyde	Acetaldehyde	Acrolein	Acetone	
Ethylene	0.35[a], 0-45[b] 0.32[c], 0.45[d]	<0.01[c]			
Propylene	0.40[a], 0.45[e]	0.4[a], 0.15-0.2[e]		0.03-0.05[e]	
1-Butene	0.45[a], 0.4[f]				0.9[a], 0.5[f]
Isobutene	0.7[a], 0.6[b] 0.3-0.45[d], 0.6[e], 0.6[f], 0.5-0.7[g]	0.01[e]		0.6[a], 0.5[d] 0.6[g], 0.25-0.4[e]	
trans-2-butene	0.35[f], 0.35[h]	1.40[a], 1.5[f], 0.9-1.2[h]			
cis-2-butene		0.9[a], 0.8-1.0[d]			1.0[b], 0.9[g]
1,3-Butadiene	0.6[a], 0.6[e], 0.5[f]	~0.01[e]	0.55[a], 0.25[i], 0.2[e]	~0.02[e]	
1-Pentene	0.55[a]				0.4[a]
2-Methyl-2-butene	0.5[a], 0.3[e]	0.75[a], 0.4-0.5[e]		0.45[a], 0.3-0.4[e]	
1,3-Pentadiene	0.65[a]		0.35[a]		1.3[a]
2-Methyl-1,3-Pentadiene	0.55[a]		0.4[a]		0.9[a]
cis-3-hexene					1.0[b], 0.9-1.0[d] 0.9-1.0[g]
2,3-Dimethyl-2-butene	0.25[a]			1.25[a], 0.8-0.9[g], 1.4[j]	
Cyclohexene	0.4[e]				
2,3-Dimethyl-1,3-butadiene	0.65[a]				1.3[a]
3-Heptene	0.8[a]				1.2[a]
Toluene					0.11[k]
p-Xylene	0.15[a]				0.26[b]
o-Xylene					0.22[b]
m-Xylene	0.15[k]				0.25[b], 0.3[k]
1,3,5-Trimethylbenzene	0.15[k]				0.6[b], 0.3[k], 0.4[l]
1,2,4,5-Tetramethylbenzene					0.45[b]

[a] Schuck and Doyle (45).

[b] Stephens and Scott (54).

[c] Altshuller and Cohen (64).

[d] Stephens et al. (65).

[e] Schuck et al. (66).

[f] Sigsby et al. (67).

[g] Stephens (57).

[h] Tuesday (68).

[i] Altshuller and Bufalini (41).

[j] Bufalini and Altshuller (69).

[k] Leighton (26).

[l] Vrbaski and Cvetanovic (70).

d. Organic Nitrates and Nitrites

Of the nitrogen-containing organics produced in irradiation studies, peroxyacetyl nitrate is clearly the most common and most abundant. Table 1-16 shows maximum yields of PAN and alkyl nitrates in irradiation studies with various olefins and alkylbenzenes. Yields of PAN were less than 0.25 mole per mole of initial hydrocarbon, with the exception of trans-2-butene and 2,3-dimethyl-2-butene, for which some yields larger than 0.5 were found. Yields of alkylnitrates were uniformly 0.25 or less.

On photooxidation of alkanes (butane, isopentane, 2-methylpentane, 3-methylhexane, 2,4-dimethylhexane, n-nonane, and methylcyclohexane), yields of peroxyacetyl nitrate and alkyl nitrates were about 1 percent or less of the initial hydrocarbon concentration, which was usually

Table 1-16. YIELDS OF PEROXYACYL NITRATES AND ALKYL NITRATES OBSERVED IN PHOTOOXIDATION OF VARIOUS OLEFINS AND ALKYLBENZENES (41)

Hydrocarbon	Highest yield, relative to initial hydrocarbon	
	PAN	Alkyl nitrate
Ethylene[a]	0	
Propylene[a,b]	0.25	
1-Butene[a]	0.11	
Isobutene[b,c]	0.23	0.10
trans-2-Butene[b]	0.51	0.15
cis-2-Butene	0.16	0.12
cis-3-Hexene[c]	0.16	0.12
trans-3-Hexene[c]	0.20	0.14
2,3-Dimethyl-2-butene[b]	0.69	0.25
p-xylene[a]	0.08	
o-Xylene[a]	0.08	
m-Xylene[a]	0.11	
1,3,5-Trimethylbenzene[a,b]	0.16	0.15
1,2,4,5-Tetramethylbenzene[a]	0.14	

[a] Stephens and Scott (54).

[b] Tuesday (56).

[c] Stephens (57).

3 ppm in these tests (48). Individual alkyl nitrates identified were methyl, ethyl, propyl, and isopropyl nitrates. Very small amounts of alkyl nitrites have been reported as products in chamber irradiations (69), and nitromethane has been found in experiments that reduced oxygen concentrations (68).

e. Minor Products

Small yields of epoxides, alcohols, esters, and peroxides from photooxidation of hydrocarbons have been reported (41). Carbon monoxide is sometimes formed in appreciable amounts (0.2 to 0.4, relative to initial hydrocarbon), (45,68), and carbon dioxide has been found in yields up to 0.25, with 2-butene as the substrate (68). These products probably have been formed in various experiments in which they were not detected, either because the necessary analysis was not done or because the concentrations were below the limits of detection of the analytical techniques used.

I. METEOROLOGICAL EFFECTS

1. General

The diurnal urban emission pattern of oxidant-forming pollutants is fairly uniform from weekday to weekday. It is apparent, therefore, that variations in the pattern of oxidant concentrations must be attributable largely to meteorological factors. Dilution of oxidants is accomplished by the same process of atmospheric turbulence and transport that affects other gaseous contaminants. The large range of values over which these parameters extend often contributes significantly to the daily and seasonal variations of observed oxidant concentrations at a particular location. Atmospheric concentrations of oxidant are also dependent upon those meteorological variables affecting oxidant formation. Some of these factors are discussed in the following sections.

2. Sunlight

Light is measured in parameters of intensity and wavelength. Intensity is the measure of the number of photons, or the concentration of light, while wavelength is the measure of how much energy each photon contains. The longer the wavelength, the less energy is contained in each photon.

Interaction of light with an NO_2 molecule to form NO and an O atom (reaction 1) is a reaction between a photon and the NO_2 molecule. Thus, the rate of NO_2 destruction, or of O atom formation, is directly proportional to the intensity of light and the concentration of NO_2. Since ozone concentration is a function of O atom concentration (reaction 2), the ozone concentration by equation 4 is likewise directly proportional to the light intensity.

NO_2 efficiently absorbs most wavelengths of light between 3000 and 6000 Å (26,71). Efficient absorption alone, however, does not guarantee that NO_2 dissociation (reaction 1) will take place. It has been determined that the dissociation of NO_2 is a function of wavelength (26,71). Over 90 percent of NO_2 molecules are photolyzed when the light wavelength is between 3000 and 3700 Å (71). Above 3700 Å, the percentage drops off rapidly; and above 4200 Å, dissociation does not occur.

Disruption of the bond between O and NO in NO_2, as necessitated in reaction 1, requires approximately 72 kilocalories of energy per mole at 25° C. This corresponds to the energy available in light at a wavelength of approximately 4000 Å. At longer wavelengths, light cannot break the NO_2 bonds because insufficient energy is available.

The wavelength at which NO_2 dissociation fails to occur is not precisely defined because the individual molecules of NO_2 do not all possess the same ground-state energy prior to absorption. Figure 1-5 shows that the transition from 100 percent to 0 percent dissociation occurs between 3700 and 4200 Å. This gradual transition indicates a variation in ground state energy of about 10 kilocalories per mole.

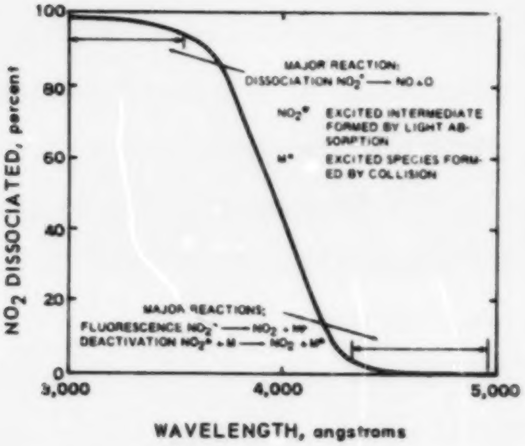

Figure 1-5. Nitrogen dioxide dissociation as a function of wavelength of light absorbed.

The relative distribution of the wavelengths of sunlight reaching the earth's surface does not vary appreciably except in the presence of absorbing species or light-scattering particles. Because some polluted atmospheres contain variable amounts of NO_2, and because NO_2 will absorb certain wavelengths of sunlight, there can be some differences in both the relative wavelength distribution and the intensity of sunlight at the earth's surface. Because ambient NO_2 concentrations are low, such effects of NO_2 absorption on wavelength distribution and intensity are likely to be small.

A reduction as high as 80 percent in the intensity of sunlight near the region of 3250 Å has been observed at the earth's surface during an intense photochemical air pollution episode (72). Since most of it had disappeared from the atmosphere at this time, NO_2 cannot be responsible for the observed decrease in light intensity. Rather, the reduction has been generally attributed to the light-scattering effect of atmospheric aerosols formed as a byproduct of the photochemical interactions of RH, NO_x, and SO_2.

Light intensity reductions of the magnitude observed would be expected to produce a substantial decrease in the rate of photochemical reactions. The actual effect, however, that is caused by aerosol diffusion is far more complex. As pointed out by Leighton (26), the available light energy is a function of height within a given polluted air mass. Within the upper half of the air mass, the available light energy will tend to be the result of aerosol scattering, and this will be substantially greater than the energy available from incident radiation alone. An opposite effect is observed in the lower half of the air mass. Thus, the formation of photochemical aerosols has the rather interesting effect of increasing the rate of photochemical reactions in the upper half of the polluted air mass and, at the same time, decreasing these rates in the lower half of the air mass. Thus, in areas where visibility reduction is severe, a less direct correlation between observed oxidant concentrations and light intensity measured at ground level would be expected. The total effect on oxidant levels of such intensity effects cannot be ascertained without quantitative data on vertical mixing within a polluted air mass.

Variations in sunlight intensity that must affect the development of oxidants are those occurring as a function of time of day, time of year, and geographical location. Maximum intensities prevail around noon, with the duration of near maximum intensities varying according to season and latitude. Cloud cover is, of course, another important factor, as is the atmospheric accumulation of light-scattering and light-absorbing pollutants.

The combination of factors of light intensity and duration somewhat controls the amount of photooxidized materials that can be formed. In the United States, the maximum noonday intensity and the duration of nearly maximum light intensity do not vary appreciably with latitude during the summer months. In the region of 3000 to 4000 Å, the maximum total intensity is 2×10^{16} photons cm^{-2} sec^{-1}, with the intensity remaining near this value for 4 to 6 hours (37). By contrast, the winter values vary from 0.7×10^{16} to 1.5×10^{16} photons cm^{-2} sec^{-1}, depending on latitude and time of nearly maximum light intensity. The amount of this time near-maximum light intensity in the winter is reduced to 2 to 4 hours (37). These times and intensities are important controlling factors in determining the severity and duration of photochemical air pollution symptoms.

3. Temperature

The temperature of a polluted air mass determines the ground-state energy of all chemical species in the system. High temperatures increase ground-state energy. In Figure 1-5, for example, the transition curve can be slightly shifted to longer wavelengths by increasing the temperature and, thus, increasing the ground-state energy of the system (71). Since most chemical reactions require addition or subtraction of energy, a temperature variation can also change the reaction rate.

Laboratory experiments have shown that a 22°C temperature rise increases the rate of NO and hydrocarbon oxidations by a factor of two (73). There is also evidence in certain systems that a temperature increase of this magnitude results in a fourfold increase in rate of oxidant production (74). These are substantial changes that can affect the concentrations of photochemical air pollution products in the atmosphere.

Quantitative estimates of the effect of ambient temperature variations upon these manifestations cannot be made at this time. The restrictive nature of the laboratory experiments and the lack of knowledge concerning the variables and reactions involved are two of the factors that prohibit other than qualitative estimates.

J. REACTANT CONCENTRATION STUDIES

1. Environmental Chamber Studies

A number of laboratory studies investigating the formation of photochemical air pollution have been carried out. The results of such studies are in agreement with atmospheric measurements and present a consistent picture. Ultraviolet irradiation of air mixtures that contain concentrations in the ppm range of certain hydrocarbons and NO or NO_2 leads to:

1. Oxidation of NO to NO_2 and then to nitric acid.
2. Reactions of NO_2 to form products such as peroxyacyl nitrates and inorganic nitrates.
3. Oxidation of hydrocarbons to form aldehydes and ketones.
4. Formation of O_3.
5. Increase in the rate of oxidation of SO_2 to SO_3 and sulfate.

Rather extensive data are available on the consumption of reactant hydrocarbons and nitrogen oxides and on the formation of products from a number of irradiated hydrocarbon-nitrogen oxide systems (41). Acceptable carbon mass balances have been obtained for the photooxidation of hydrocarbons such as ethylene, propylene (40), 1-butene (45), *trans*-2-butene (68), *n*-butane (48), and pentanes (48). Major products vary from system to system and include formaldehyde, acetaldehyde, one or more other aldehydes or ketones, carbon monoxide, carbon dioxide, and peroxyacyl nitrates (41); other important products include ozone, nitrogen dioxide, and inorganic nitrates. Acceptable nitrogen balances have been obtained from irradiations of mixtures containing hydrocarbons such as ethylene and 1,3-butadiene, which produce little if any peroxyacyl nitrates. Acceptable carbon and nitrogen balances have not been reported from the irradiation of nitrogen oxides in combination with higher-molecular-weight hydrocarbons, such as the alkylbenzenes.

Several studies have been made of ultraviolet-irradiated automobile-exhaust systems (75-80). Ozone, nitrogen dioxide, formaldehyde and other aldehydes, and peroxyacyl nitrates have been identified as products and measured. As expected, the same types of products are formed as in the irradiation of simpler mixtures. From the results of one study, the formation of aldehydes can be estimated to account for about 30 percent of the carbon atoms consumed from the hydrocarbons.

In addition to the product-yield studies, much attention has been directed to the quantification of reaction dynamics. It was realized at an early date, however, that rates of reactions and concentrations of products are not strictly proportional to the initial concentrations of reactants. Haagen-Smit (75) first noted this effect during the irradiation of flasks containing hydrocarbons and NO_x in air. In view of the previous discussion concerning the complex hydrocarbon interaction with NO_2 photolysis, such nonlinear effects are not unexpected. Since concentration ratios, as well as absolute concentrations, of the primary reactants affect oxidant concentrations and rates of formation, a summary of the available information on these topics is required in order to appreciate oxidant variations in polluted atmospheres. Nevertheless, in discussing these effects, it must be remembered that all of the data on this subject have been generated in experimental systems that only approximate ambient atmospheres. Because of the limitations of atmospheric data, researchers have been unable to show as yet that concentration ratio effects are unequivocably operating in the real atmosphere. Still, the implications demand an examination of the experimental data.

Subsequent to the work of Haagen-Smit, several investigators have attempted to quantify the effect of changes in reactant concentrations and ratios (40,45,76,78). In one sense, all the studies could be deemed successful in that each laboratory was able to confirm and quantify the effects. There appears to be only a qualitative agreement, however, when one study is compared with another, and there is no general agreement concerning the meaning of the results when they are extrapolated to the atmosphere. As indicated, direct atmospheric data on this subject have not as yet been obtained. A major stumbling block is the lack of air monitoring data on atmospheric concentrations of reactive hydrocarbons. Such a lack means that the applicable concentrations or ratios in the real atmosphere cannot be clearly specified at this time.

Examples of the relationships observed experimentally between reactant and oxidant concentrations are shown in Figures 1-6 through 1-9. It should be noted that the data used for Figures 1-6 and 1-8 came from studies in which mixtures containing propylene as the hydrocarbon were irradiated, while the data used for Figures 1-7 and 1-9 came from irradiated mixtures containing auto exhaust as the hydrocarbon source. In spite of many differences in choice of initial reactants and experimental design, the qualitative agreement in these four studies is rather striking. All studies show a similar dependence of oxidant on hydrocarbon concentrations. All studies further reveal a noticeable dependence of oxidant upon NO_x levels. From the previous discussion, it is not unrealistic to envision the complex NO_x effect as the direct result of the competition between the reactions resulting in oxidant formation and destruction.

K. FUTURE RESEARCH

In any evaluation of laboratory data on photochemical air pollution, it should be emphasized that environmental chamber experi-

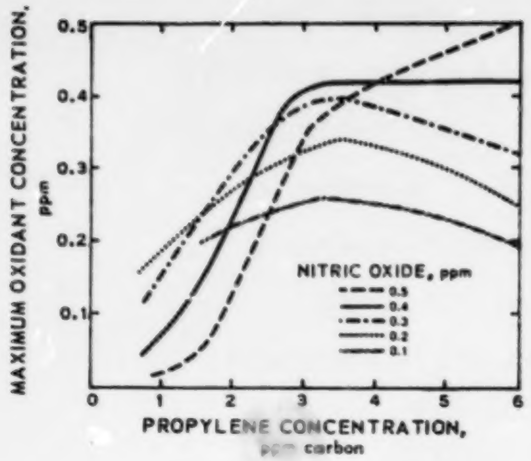

Figure 1-6. Maximum oxidant concentration as a function of propylene and nitric oxide reactant concentrations during 2-hour dynamic irradiation (40).

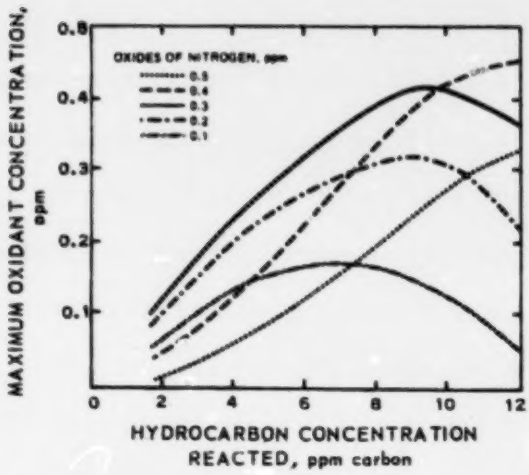

Figure 1-7. Maximum oxidant concentration as a function of hydrocarbon and nitrogen oxide concentrations reacted during 1-hour dynamic irradiation of automobile exhaust mixtures (81).

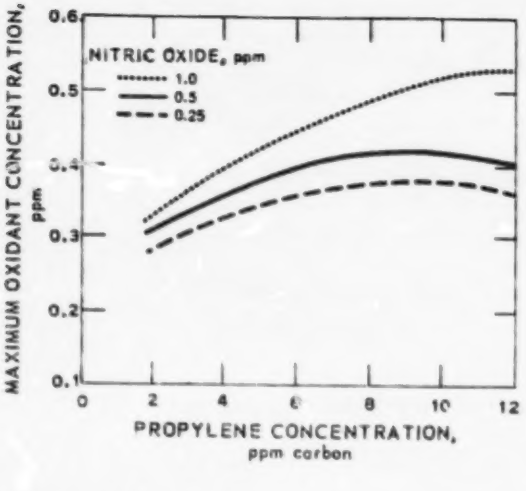

Figure 1-8. Maximum oxidant concentration as a function of propylene and nitric oxide reactant concentrations during static irradiations (78).

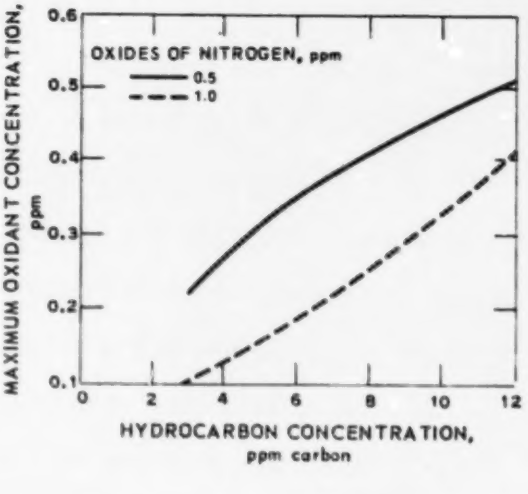

Figure 1-9. Maximum oxidant concentration as a function of hydrocarbon and nitrogen oxide reactant concentrations during 2-hour dynamic irradiation of automobile exhaust mixtures (76).

ments are relatively simple when compared to the highly complex and variable situations occurring in the atmosphere. The fact that various laboratory investigators have obtained significantly different quantitative results illustrates this contrast. Such limited findings can only be applied to the ambient atmosphere in a suggestive and qualitative manner. There is an apparent need for more reliable and applicable quantitative information derived from direct atmospheric observations, as well as for refinement in the results obtained from irradiation chamber studies.

Relevant monitoring data in more detail are needed on reactant concentrations and meteorological factors. Because these elements control the size, ventilation rate, and mixing characteristics of polluted air masses, they determine to a large extent pollutant concentrations and photochemical reaction products.

The need for all types of basic irradiation chamber experimentation remains acute. A partial listing of such needed experiments is:

1. Kinetic measurements of the rates of reaction of hydroxyl radicals with various hydrocarbons. The observed overall rates of hydrocarbon consumption are not explained by reactions with atomic oxygen and ozone. The most likely intermediate species contributing to these reactions are hydroxyl radicals.

2. The relationship of oxidants, oxygenated hydrocarbons, organic nitrates, and other possible substances to such factors as reactant concentrations, reactant concentration ratio, temperature, light intensity, and water vapor. While the products accounting for most of the carbon atoms from lower-molecular-weight paraffinic hydrocarbons

and from olefinic hydrocarbons have been measured, the results of carbon balances from the reactions of alkylbenzenes and higher-molecular-weight paraffins are poor. Although the presence of dicarbonyl compounds from alkylbenzenes has been reported, quantitative measurements are lacking. Peroxybenzoyl nitrate, a new and minor product from the reaction of certain alkylbenzenes, is a potent eye irritant that has recently been identified in this type of research.

3. The relationship of aerosol formation to a variety of reaction parameters, including water vapor content. Studies of the role of sulfur dioxide, sulfuric acid, sulfates, nitric acid, nitrates, and organics in aerosol formation should also be extended. The role of organics, especially, should be studied, inasmuch as the composition of the organic fraction of aerosols is still unknown.

4. More measurements on the yields of organic and inorganic nitrates from a variety of hydrocarbon-nitrogen oxide systems. The formation of inorganic nitrates has been shown to occur at an appreciable rate and as a direct result of the photooxidation of hydrocarbons in the presence of nitrogen oxides.

5. Additional investigation of the importance of other primary absorbers or species made reactive by energy transfer. Such species include the products of aldehydes and singlet oxygen.

Even more urgently, detailed investigations of the atmospheric reactions in several representative urban atmospheres are needed. The type of information required includes:

1. Reactant and product composition obtained from the irradiation of atmospheric samples with solar radiation at atmospheric temperatures.

Such results can be used to verify kinetic and product composition results obtained in the laboratory. Such measurements have been made in a limited group of experiments on samples collected in Los Angeles.

2. Detailed results on the composition of ground-level samples and samples obtained aloft. Coordinated vertical distribution measurements along trajectories are needed to trace the reaction history of air masses. Oxidant concentrations should be related to hydrocarbon-nitrogen oxide concentration levels. Such measurements should also emphasize clarification of the importance of various species identified in laboratory irradiations but not in the atmosphere, such as peroxybenzoyl nitrate, hydrogen peroxide, alkylhydroperoxides, and dicarbonyls.

3. Measurement techniques to estimate the actual concentrations of radical intermediate species in urban atmospheres.

4. Measurement techniques for obtaining hourly concentrations of total particulates, submicron particulates, sulfate, nitrate, lead, and other particulate species and fractions. Although a considerable body of measurements are available on diurnal variations in gaseous pollutants, comparable diurnal measurements of particulates are not available.

L. SUMMARY

The atmospheric reactions leading to formation of the photochemical oxidants, ozone and peroxyacyl nitrates, are reasonably well understood broadly if not in detail. It is known that certain hydrocarbon pollutants interact with the photolytic cycle of nitrogen dioxide and that, as a result, the hydrocarbons are oxidized to form various products. The product mixtures contain substantial concentrations of ozone, as well as such hydrocarbon products as aldehydes, ketones, and peroxyacyl nitrates.

Absorption of sunlight by nitrogen dioxide in the region of 3000 to 4000 Å results in dissociation of the nitrogen dioxide into nitric oxide and an oxygen atom. These oxygen atoms react principally with the oxygen in air to form ozone. A small portion of the oxygen atoms and ozone also react with certain hydrocarbons to form free-radical intermediates, as well as various products. In some unknown manner, these free-radical intermediates compete with ozone for nitric oxide. One result is the very rapid oxidation of nitric oxide to nitrogen dioxide and a concomitant increase in the concentration of ozone.

Experimentally, this photochemical system can be reproduced in the laboratory, and data can be obtained that relate oxidant concentrations to the concentrations and types of both hydrocarbons and oxides of nitrogen. For various reasons, however, these results cannot be extrapolated to the atmosphere in a quantitative manner. Because of the complexity of the atmospheric mixture and the necessary use of nonspecific monitoring techniques, demonstration of the relationship of hydrocarbon and oxidant concentrations in the atmosphere is somewhat limited. A more precise examination of the parameters affecting atmospheric oxidant concentrations awaits the application of statistical and modeling techniques as well as improvements in measuring methods.

The hydrocarbons of interest in this document are those that may be encountered in the gas phase in urban atmospheres. In this context, hydrocarbons having a carbon number greater than about 12 are generally not encountered in the atmosphere in sufficiently high gaseous concentrations to be of concern.

Most natural sources of hydrocarbon emissions are biological in nature. A conservative estimate made for the worldwide natural production rate of methane is 2.7×10^8 MT per year. A similar estimate of 4.0×10^8 MT per year was made for volatile terpenes and isoprenes. Estimates of hydrocarbon background concentrations have been made by several authors using a variety of techniques. It appears that nonurban air contains from 0.7 to 1.0 mg/m^3 (1.0 to 1.5 ppm) methane and less than 0.1 ppm each of other hydrocarbons.

Total nationwide technological emissions of hydrocarbons and related organic compounds have been estimated and transportation has been found to be the largest source category.

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CHAPTER 2

ATMOSPHERIC CONCENTRATIONS

A. HYDROCARBONS AND RELATED COMPOUNDS

Atmospheric hydrocarbon concentrations are of interest in that they relate to the production of photochemical oxidants. Several factors should be considered concerning atmospheric hydrocarbon data. There is great variation in the respective tendencies of different hydrocarbons to participate in photochemical reactions. The simpler detection methods are likely to respond most strongly to methane and other alkanes. Methane is often more abundant in the atmosphere than all other hydrocarbons combined. There is, in fact, apparently a "geophysical" minimum concentration of methane, present worldwide, of about 0.7 to 1.0 mg/m^3 (1.0 to 1.5 ppm) (1-3). Methane, though consistently present and detected, does not enter into photochemical reactions and therefore is not of interest in relation to photochemical reactions. It is important, then, in assessing photochemical air pollution to be able to discriminate between methane and other more reactive hydrocarbons. Unfortunately, this type of analysis is expensive and time-consuming, so that such data are not abundant.

The importance of considering the location of a particular monitoring site when interpreting ambient hydrocarbon levels should also be pointed out. The ambient concentrations that are observed at a given site are influenced greatly by a number of factors. The two most important of these factors are 1) the specific site location with respect to the principal hydrocarbon emission sources, and 2) the meteorological factors that will greatly influence the specific ambient concentration levels observed. A proper analysis of these two factors will often place into perspective and facilitate interpretation of the ambient concentration levels that are observed.

1. Hydrocarbons

Although on occasion all other hydrocarbon concentrations drop to unmeasurably low levels, methane does not. Numerous measurements suggest a worldwide minimum methane concentration of about 0.7 to 1.0 mg/m^3 (1.0 to 1.5 ppm) (3). In inhabited areas, methane levels are often much higher; values of 4 mg/m^3 (6 ppm) or more have been observed (4).

Ratios of nonmethane hydrocarbons (as carbon) to methane have been estimated for urban areas, after subtracting 1 ppm from methane values to allow for estimated biogenic background levels (4). The nonmethane/methane hydrocarbon ratios for several weeks averaged 0.6 in Cincinnati, Ohio, and 1.9 in Los Angeles, although methane values were similar (Figure 2-1.) The higher Los Angeles ratios may possibly result from local differences in traffic density and solvent losses.

a. Hydrocarbons in Ambient Air

Table 2-1 lists the individual hydrocarbons detected in samples of urban air by gas chromatographic analysis in several investigations in the U. S. (5-7). The distribution of the 56 compounds detected was: 17 alkanes, 23 alkenes (including two alkadienes), 2 alkynes, 10 aromatics, 3 cycloalkanes, and 1 cycloalkene. The length of this list is limited only by the sensitivity of the

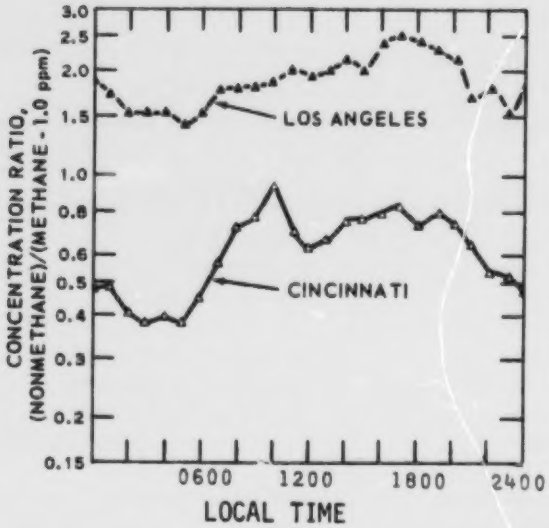

Figure 2-1. Concentration ratios for non-methane hydrocarbons/methane in Los Angeles (213 hours during October and November, 1964) and Cincinnati (574 hours during September 1964), with 655 μg/m^3 (1 ppm) methane deducted to correct for estimated background biospheric concentration (4).

Table 2-1. SOME HYDROCARBONS IDENTIFIED IN AMBIENT AIR IN THE UNITED STATES

Carbon number	Class	Compound	Reference
1	Alkane	Methane	6
2	Alkane	Ethane	5,6
2	Alkene	Ethylene	5,6
2	Alkyne	Acetylene	5,6
3	Alkane	Propane	5,6
3	Alkene	Propylene	5-7
3	Alkene	Propadiene	6
3	Alkyne	Methylacetylene	6
4	Alkane	Butane	5,6
4	Alkane	Isobutane	5,6
4	Alkene	1-Butene	5,6
4	Alkene	*cis*-2-Butene	5,6
4	Alkene	*trans*-2-Butene	5,6
4	Alkene	Isobutene	5,6
4	Alkene	1,3-Butadiene	5,6
5	Alkane	Pentane	5,6

Table 2-1 (Continued). SOME HYDROCARBONS IDENTIFIED IN AMBIENT AIR IN THE UNITED STATES

Carbon number	Class	Compound	Reference
5	Alkane	Isopentane	5,6
5	Alkene	1-Pentene	5,6
5	Alkene	*cis*-2-Pentene	5,6
5	Alkene	*trans*-2-Pentene	5
5	Alkene	2-Methyl-1-butene	5-7
5	Alkene	2-Methyl-2-butene	5-7
5	Alkene	3-Methyl-1-butene	5
5	Alkene	2-Methyl-1,3-butadiene	5
5	Cycloalkane	Cyclopentane	5,6
5	Cycloalkane	Cyclopentene	5,6
6	Alkane	Hexane	5,6
6	Alkene	2-Methylpentane	5,6
6	Alkane	3-Methylpentane	5,6
6	Alkane	2,2-Dimethylbutane	5,6
6	Alkane	2,3-Dimethylbutane	5,6
6	Alkene	1-Hexene	6
6	Alkene	*cis*-2-Hexene	6
6	Alkene	*trans*-2-Hexene	6
6	Alkene	*cis*-3-Hexene	6
6	Alkene	*trans*-3-Hexene	6
6	Alkene	2-Methyl-1-pentene	6
6	Alkene	4-Methyl-1-pentene	6
6	Alkene	4-Methyl-2-pentene	6
6	Aromatic	Benzene	6
6	Cycloalkane	Cyclohexane	6
6	Cycloakane	Methylcyclopentane	6
7	Alkane	2-Methylhexane	6
7	Alkane	3-Methylhexane	6
7	Alkane	2,3-Dimethylpentane	6
7	Alkane	2,4-Dimethylpentane	6
7	Aromatic	Toluene	6,7
8	Alkane	2,2,4-Trimethylpentane	6
	Aromatic	*o*-Xylene	7
		m-Xylene	7
		p-Xylene	7
9	Aromatic	*m*-Ethyltoluene	7
		p-Ethyltoluene	7
		1,2,4-Trimethylbenzene	7
		1,3,5-Trimethylbenzene	7
10	Aromatic	*sec*-Butylbenzene	7

analytical methods, and it is certain that many additional hydrocarbon compounds are actually present in urban air. Especially at the higher carbon numbers, the complexity of the chromatographic records becomes so great that the effort to interpret them in terms of individual compounds may be insufficiently rewarding. The listing in Table 2-1 of compounds with carbon numbers of 7 and higher must be considered only a partial list.

All the hydrocarbons above carbon number 4 listed in Table 2-1 are found in gasoline (8). These and the lower alkenes and acetylenes are also found in automobile exhaust gases. The lower alkanes (methane, ethane, and propane) occur in only trace amounts in auto exhaust gases, but are ordinary constituents of natural gas. Stephens and Burleson (5) have reported that the hydrocarbon composition of their ambient air samples resembled that of auto exhaust gases with an addition of natural gas and gasoline vapor. However, samples taken in industrial areas and from near the smoke plume from a brush fire have shown distinctive differences in composition, which reasonably can be attributed to these particular sources. Special studies such as these can be very useful in identifying the importance of various sources as contributors to the pollution of air by hydrocarbons.

Grab and Grab (9) have identified some 108 hydrocarbon compounds with carbon numbers of 6 to 10 in the urban atmosphere of Zurich. Table 2-2 presents their listing.

b. Diurnal Variation

Figures 2-2 and 2-3 show diurnal patterns for nonmethane hydrocarbons in several U. S. cities, averaged over several

Table 2-2. SUBSTANCES IDENTIFIED IN AMBIENT AIR IN ZURICH (9)

No.	Compound	No.	Compound
1	*a*-Methylhexane	28	Isoundecane
2	*n*-Heptane	29	Isoundecane
3	Isooctane	30	Isoundecane
4	Benzene	31	Isoundecane
5	*2*-Methylheptane	32	Isoundecane
6	*n*-Octane	33	Isoundecane
7	Isononane	34	Isoundecane
8	Isononane	35	*n*-Propylbenzene
9	n-Nonane	36	*n*-Undecane
10	Toluene	37	1-Ethyl-4-methylbenzene
11	Isodecane	38	1-Ethyl-3-methylbenzene
12	Tetrachloroethylene[a]	39	Limonene[a]
13	Isodecane	40	1,3,5-Trimethylbenzene
14	Isodecane	41	Isododecane
15	Isodecane	42	1-Ethyl-2-methylbenzene
16	Isodecane	43	Isododecane
17	Isodecane	44	1,2,4-Trimethylbenzene
18	Isodecane	45	Octenal[a]
19	Isodecane	46	1-Isopropyl-4-methylbenzene
20	Isodecane[a]	47	Isododecane
21	*n*-Decane	48	Isododecane
22	Ethylbenzene	49	Isododecane
23	1,4-Dimethylbenzene	50	*n*-Butylbenzene
24	Isoundecane	51	Propylmethylbenzene
25	Isoundecane	52	*sec*-Butylbenzene
26	1,3-Dimethylbenzene	53	1,2,3-Trimethylbenzene
27	1,2-Dimethylbenzene	54	Isotridecane

[a] Reacts with sulfuric acid.

Table 2-2 (Continued). SUBSTANCES IDENTIFIED IN AMBIENT AIR IN ZURICH (9)

No.	Compound	No.	Compound
55	n-Dodecane	82	3-Methylbenzaldehyde[a]
56	Ethyldimethylbenzene	83	Isotetradecane
57	Propylmethylbenzene	84	n-Tetradecane
58	Ethyldimethylbenzene	85	Acetophenone[a]
59	Isopropylmethylbenzene	86	Alcohol, $C_{10}H_{20}O$[a]
60	Ethyldimethylbenzene	87	Alcohol, $C_{10}H_{18}O$[a]
61	Isopropylmethylbenzene	88	2-Methylbenzaldehyde[a]
62	Dichlorobenzene[a]	89	Naphthalene
63	Isotridecane	90	n-Pentadecane
64	Isotridecane	91	Dimethylbenzaldehyde[a]
65	Ethyldimethylbenzene	92	2-Methylnaphthalene
66	C_4-substituted benzene	93	n-Hexadecane
67	C_5-substituted benzene	94	1-Methylnaphthalene
68	1,2,4,5-Tetramethylbenzene	95	Benzothiazole[a]
69	Benzaldehyde[a]	96	n-Heptadecane
70	Benzofuran[a]	97	Dimethylnaphthalene
71	1,2,3,5-Tetramethylbenzene	98	Diphenyl
72	n-Tridecane	99	2,6-Dimethylnaphthalene
73	C_5-substituted benzene	100	1,6-Dimethylnaphthalene
74	Methylindane	101	1,8-Dimethylnaphthalene
75	C_5-substituted benzene	102	n-Octadecane
76	1,2,3,4-Tetramethylbenzene	103	Methyldiphenyl
77	Methylindane	104	Acenaphthene
78	Isotetradecane	105	n-Nonadecane
79	Isotetradecane	106	Dibenzofuran[a]
80	C_5-substituted benzene	107	Fluorene
81	4-Methylbenzaldehyde[a]	108	n-Eicosane

[a] Reacts with sulfuric acid.

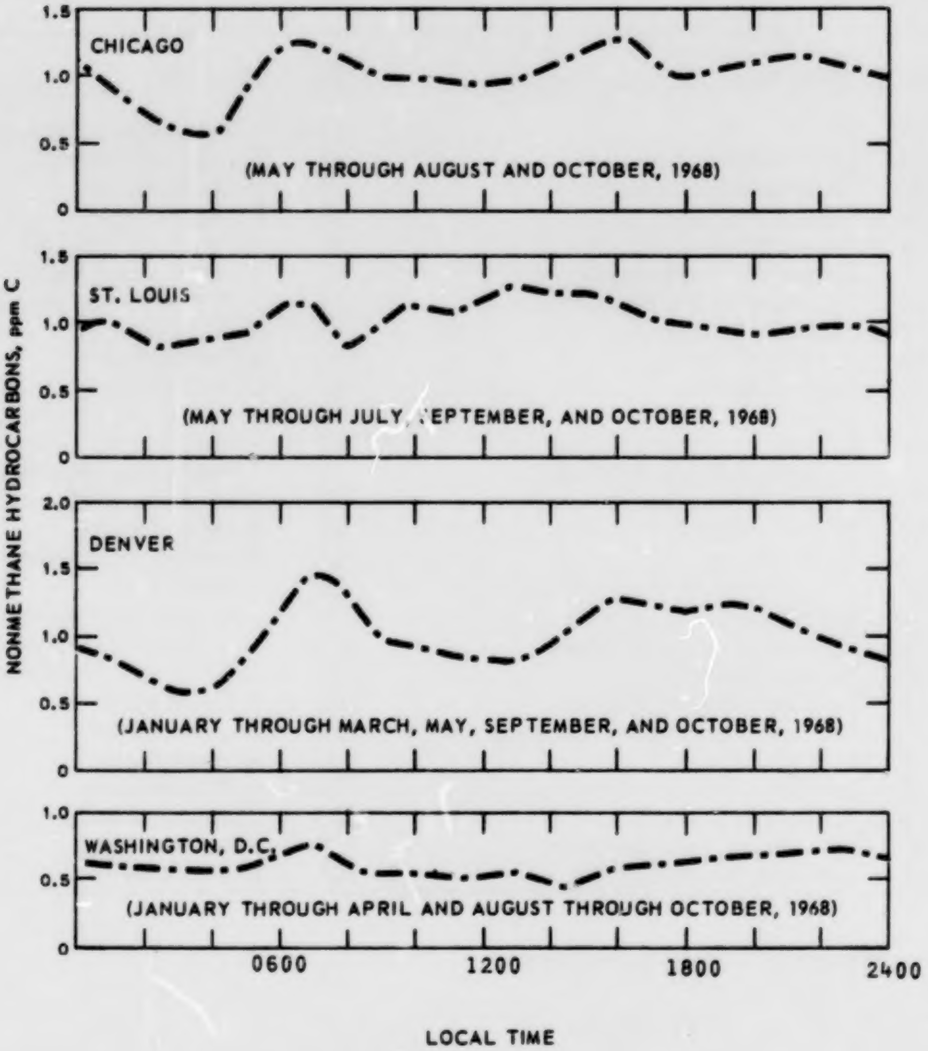

Figure 2-2. Nonmethane hydrocarbons by flame ionization analyzer, averaged by hour of day over several months for various cities

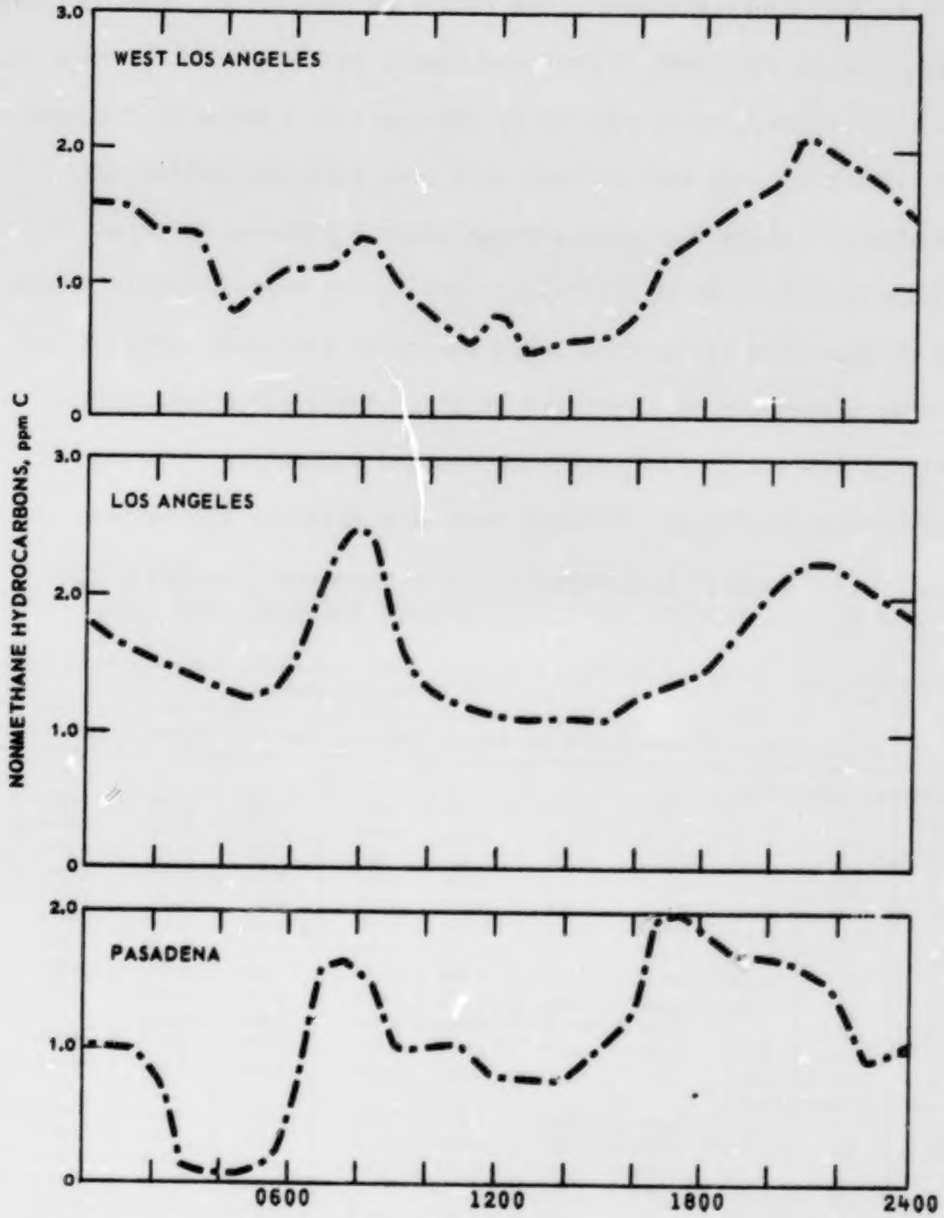

Figure 2-3. Nonmethane hydrocarbons determined by flame ionization analyzer and averaged by hour of day for three Los Angeles county sites, October 1966 through February 1967 (11)

months (10,11). In most of these, the maximum concentrations at 0600 to 0800 hours are mainly the result of the morning commuter traffic rush. The morning peak is most pronounced in Denver and Los Angeles, where the automobile is especially important as a means of transportation. Even within a large metropolitan area, there may be considerable variation. Figure 2-3 shows average diurnal patterns for non-methane hydrocarbons at three locations in Los Angeles County. There are considerable differences among the three patterns, although the morning maxima are still evident in all. Table 2-3 gives diurnal patterns for the C_2 to C_4 hydrocarbons and isopentane, showing the hour-by-hour variations averaged over some weeks in the Los Angeles

Table 2-3. AVERAGE ATMOSPHERIC LIGHT HYDROCARBON CONCENTRATIONS BY HOUR, LOS ANGELES, SEPTEMBER THROUGH NOVEMBER, 1967 (12)

Local time	No. of days averaged	Hydrocarbon, ppm C								
		Ethane	Ethylene	Acetylene	Propane	Propylene	Isobutane	*n*-Butane	Butylenes	Isopentane
Downtown Los Angeles (DOLA)										
0500	27	0.140	0.031	0.077	0.113	0.025	0.053	0.176	0.032	0.177
0600	28	0.140	0.049	0.112	0.113	0.036	0.061	0.200	0.034	0.224
0700	28	0.151	0.122	0.234	0.125	0.064	0.074	0.300	0.056	0.335
0800	31	0.145	0.102	0.212	0.126	0.054	0.075	0.304	0.049	0.346
0900	15	0.107	0.063	0.141	0.098	0.032	0.063	0.209	0.028	0.249
1000	33	0.126	0.067	0.152	0.118	0.038	0.064	0.260	0.034	0.303
1100	12	0.094	0.039	0.106	0.080	0.025	0.048	0.180	0.021	0.200
1200	32	0.106	0.039	0.101	0.108	0.020	0.063	0.200	0.015	0.199
1300	16	0.076	0.020	0.068	0.078	0.013	0.044	0.149	0.014	0.162
1400	38	0.094	0.026	0.074	0.097	0.017	0.057	0.156	0.019	0.155
1500	16	0.090	0.026	0.079	0.101	0.016	0.050	0.132	0.020	0.144
1600	34	0.075	0.036	0.095	0.081	0.024	0.046	0.139	0.021	0.141
East San Gabriel Valley (Azusa)										
0500	32	0.143	0.029	0.060	0.087	0.013	0.036	0.101	0.014	0.104
0600	33	0.128	0.017	0.065	0.076	0.015	0.031	0.098	0.012	0.103
0700	30	0.180	0.030	0.103	0.102	0.024	0.041	0.136	0.018	0.144
0800	32	0.118	0.037	0.101	0.084	0.020	0.044	0.149	0.018	0.160
0900	18	0.093	0.031	0.079	0.065	0.015	0.036	0.114	0.015	0.114
1000	32	0.086	0.024	0.069	0.066	0.019	0.040	0.132	0.015	0.136
1100	18	0.090	0.021	0.057	0.062	0.011	0.037	0.107	0.010	0.101
1200	30	0.089	0.015	0.050	0.058	0.010	0.040	0.098	0.009	0.093
1300	16	0.111	0.016	0.066	0.067	0.018	0.046	0.121	0.017	0.111
1400	42	0.097	0.018	0.064	0.075	0.017	0.050	0.121	0.018	0.117
1500	16	0.092	0.020	0.065	0.075	0.015	0.081	0.175	0.015	0.125
1600	37	0.079	0.021	0.058	0.076	0.014	0.041	0.122	0.014	0.123

smog season. All species listed reached a maximum in the morning, then declined through the midday (12). Table 2-4 shows similar data from The Hague, The Netherlands (13).

c. Seasonal Variation

Necessary data are not now available for the assessment of seasonal or annual variations in nonmethane hydrocarbon levels. In most instances, total hydrocarbons consist mostly of methane, which is, as mentioned previously, photochemically inert. Total hydrocarbon concentrations are tabulated for 17 California cities in Table 2-5 (14). Note that 14 of these cities show their highest hydrocarbon concentrations (averages of maximum hourly averages) in October or November. This is presumably a consequence of the generally similar meteorological conditions along the California coast. Cities elsewhere would be expected to show other patterns, dependent on their particular meteorology. Investigators have reported total hydrocarbon averages of 4.0 ppm, 4.3 ppm, and 3.3 ppm for September, October, and November respectively, in downtown Frankfurt/Main (15). The maximum 30 minute average measurement of total hydrocarbons in the city of Berlin, Germany in 1967 and 1968 was 17.2 ppm (42).

d. Secondary Contaminants

The photochemical interaction of organic materials in the atmosphere with oxides of nitrogen leads to numerous products, including oxidants such as ozone, carbonyl compounds, and peroxyacyl nitrates. Atmospheric aerosols and particulate matter also play a part in the formation of secondary contaminants. These materials are available for the attachment of the various oxidation products of hydrocarbon species.

Table 2-4. AVERAGE ATMOSPHERIC LIGHT HYDROCARBON CONCENTRATIONS BY HOUR, THE HAGUE, SEPTEMBER, 1973 (13)

Local time	No. of days averaged	Hydrocarbon, ppm									
		Ethane	Ethylene	Acetylene	Propene	Propylene	Isobutane	n-Butane	Isopentane	n-Pentane	Methane
00.00-01.00	26	0.012	0.018	0.022	0.009	0.005	0.005	0.012	0.011	0.005	2.3
01.00-02.00	26	0.014	0.015	0.017	0.010	0.004	0.004	0.009	0.009	0.004	2.4
02.00-03.00	26	0.013	0.010	0.009	0.009	0.003	0.003	0.007	0.005	0.003	2.4
03.00-04.00	26	0.013	0.008	0.007	0.007	0.002	0.002	0.005	0.004	0.002	2.4
04.00-05.00	26	0.013	0.008	0.006	0.007	0.001	0.002	0.005	0.004	0.002	2.2
05.00-06.00	26	0.013	0.007	0.005	0.007	0.001	0.002	0.004	0.003	0.002	2.4
06.00-07.00	26	0.013	0.008	0.006	0.009	0.002	0.002	0.006	0.003	0.002	2.4
07.00-08.00	26	0.014	0.013	0.012	0.013	0.004	0.004	0.010	0.007	0.003	2.4
08.00-09.00	26	0.014	0.025	0.028	0.012	0.006	0.006	0.015	0.013	0.006	2.3
09.00-10.00	26	0.014	0.021	0.021	0.015	0.006	0.007	0.016	0.013	0.006	2.3
10.00-11.00	26	0.010	0.018	0.020	0.011	0.006	0.006	0.014	0.012	0.006	2.1
11.00-12.00	26	0.008	0.015	0.018	0.009	0.004	0.006	0.013	0.012	0.005	2.0
12.00-13.00	26	0.007	0.014	0.018	0.007	0.003	0.005	0.012	0.010	0.004	2.0
13.00-14.00	26	0.007	0.014	0.018	0.008	0.003	0.005	0.012	0.011	0.005	1.9
14.00-15.00	26	0.007	0.015	0.020	0.007	0.003	0.005	0.012	0.012	0.005	1.9
15.00-16.00	26	0.007	0.015	0.022	0.006	0.003	0.004	0.011	0.011	0.005	1.9
16.00-17.00	26	0.007	0.020	0.032	0.006	0.004	0.005	0.013	0.014	0.006	1.9
17.00-18.00	26	0.008	0.029	0.051	0.007	0.006	0.007	0.016	0.018	0.008	2.0
18.00-19.00	26	0.008	0.021	0.031	0.006	0.005	0.006	0.016	0.016	0.007	2.0
19.00-20.00	26	0.009	0.024	0.037	0.007	0.005	0.007	0.018	0.020	0.009	2.0
20.00-21.00	26	0.010	0.023	0.034	0.008	0.005	0.007	0.018	0.019	0.008	2.1
21.00-22.00	26	0.011	0.019	0.027	0.008	0.005	0.005	0.014	0.015	0.007	2.2
22.00-23.00	26	0.011	0.019	0.024	0.007	0.004	0.004	0.012	0.012	0.006	2.2
23.00-24.00	26	0.011	0.020	0.026	0.007	0.004	0.005	0.012	0.013	0.005	2.2

Table 2-5. MEAN OF DAILY MAXIMUM HOUR AVERAGE TOTAL HYDROCARBON CONCENTRATIONS (as ppm methane), 17 CALIFORNIA CITIES, 1968-1969 (14)

	1968							1969				
	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	Apr.	May
San Francisco Bay Area												
Richmond	5	6	4	6	9[a]	8	9[a]	8	8	7	6	4
San Rafael	4	4	5	6	13[a]	10	9	6	6	8	6	6
San Francisco	5	4	N.A.[b]	5	7[a]	6	6	5	5	5	5	4
Redwood City	N.A.	12	14	16[a]	13	11	9	7	6	7	5	5
San Jose	4	4	4	6	8[a]	8[a]	7	7	5	6	6	5
Central Valley												
Fresno	3	3	3	4	6[a]	5	5	4	4	5	3	3
Bakersfield	7	7	8	10[a]	10[a]	9	10[a]	7	8	7	6	6
Sacramento	4	4	5	5	10[a]	7	7	6	5	7	4	4
Stockton	3	4	4	5	6[a]	4	5	4	3	5	4	4
Central Coast												
Salinas	3	2	3	3	3	4[a]	4[a]	3	3	3	2	N.A.
Monterey	3	2	3	3	4[a]	4[a]	4[a]	4[a]	4[a]	3	3	3
Southern California												
Los Angeles (downtown)	4	5	4	5	6	7[a]	7[a]	6	4	5	4	3
Azusa	4	4	5	5	6[a]	5	5	4	4	5	5	5
Anaheim	4	5	5	6	8	10[a]	10[a]	7	7	7	5	3
Riverside	5	5	7	6	7	7	8	7	9[a]	8	4	4
San Bernardino	4	5	5	6[a]	6[a]	6[a]	6[a]	5	N.A.	6[a]	5	4
San Diego	3	N.A.	4	5	7	7	9[a]	7	6	6	5	3

[a] Highest mean concentration for 12-month period.

[b] N.A. = Not available.

2. Aldehydes

Of all the principal reaction products formed in the atmosphere by photochemical processes, the aldehydes are among the most poorly quantified. A maximum of 39 $\mu g/m^3$ (0.032 ppm) formaldehyde was measured in a street with high traffic density in Berlin, Germany in 1967 (43).

The ambient concentrations of aliphatic aldehydes were measured in Rotterdam during the period January through March 1973. The average 24-hour concentration was 5.2 $\mu g/m^3$ and the 0800 to 1600 hour average was 8.4 $\mu g/m^3$ (16).

Most of the available data are from Los Angeles, where from 1951 through 1957 aldehydes were regularly measured by manual techniques (17). Total aldehydes was determined by the bisulfite procedure (18) and reported as formaldehyde; formaldehyde was determined by the chromotrophic acid procedure (19).

A study in Los Angeles County from July to November, 1960, provided additional data on total aldehydes, formaldehyde, and acrolein (20). Total-aldehyde concentrations ranged up to 0.36 ppm for a 10-minute sample, but formaldehyde did not exceed 130 $\mu g/m^3$ (0.10 ppm). Typical aldehyde concentrations were near 0.10 ppm on many days. The maximum acrolein values was 25.2 $\mu g/m^3$ (0.011 ppm), with most values being less than half that amount.

Diurnal variations in aldehydes are apparent for analytical data obtained in Los Angeles from September to November, 1961 (21). These data are shown in Table 2-6. The concentrations show an early rise, a broad plateau or maximum through most of the day, and a decrease in the afternoon. It is apparent that formaldehyde contributes the bulk of the aliphatic aldehyde concentration, whereas the acrolein concentration is less than one-tenth that of formaldehyde.

In these few available studies of ambient levels of aldehydes, the ratio of formaldehyde to total aldehydes showed no consistent value, ranging from about 10 percent in one study (20) to 90 percent in another (22). This inconsistency may be attributable in part to the use of different methods for analyzing aldehydes.

3. Aerosols

Table 2-6. AVERAGE ALDEHYDE CONCENTRATIONS BY HOUR IN LOS ANGELES, SEPTEMBER 25 THROUGH NOVEMBER 15, 1961 (21)

Sampling time	Formaldehyde		Acrolein	
	Number of days	Average concentration, ppm	Number of days	Average concentration, ppm
0700	7	0.041	2	0.007
0800	18	0.043	3	0.009
0900	21	0.045	3	0.009
1000	28	0.044	5	0.008
1100	27	0.051	5	0.008
1200	23	0.044	3	0.005
1300	25	0.041	7	0.008
1400	27	0.034	5	0.007
1500	25	0.026	4	0.004
1600	15	0.019	5	0.004

Cities in the United States with a population greater than 400,000 have an average atmospheric particulate burden of about 120 $\mu g/m^3$ (23). Some particulate matter is organic and some inorganic. Of the national U. S. average concentration of suspended particulates from 1960 through 1965, 6.5 percent consisted of benzene-soluble particulates; the total organic content might be several times this, depending on the geographic origin of the suspended particulates.

B. PHOTOCHEMICAL OXIDANTS

1. Introduction

This section is concerned with the measured levels of oxidants in urban atmospheres and some of the factors that affect these levels. It is intended to be a straightforward presentation of atmospheric measurements and the interpretation of those measurements. The only point likely to cause confusion is the various terms used to describe oxidant concentrations. Two of these terms, "oxidant" and "total oxidant," are used to describe the "net" oxidizing ability of the air sampled. Use of these nonspecific terms is required because the common method of measurement does not differentiate between oxidizing or reducing agents in all cases. The terms "corrected oxidant" or "adjusted oxidant" are used to indicate that the measurements have been corrected for certain known responses caused by gases other than ozone or peroxyacetyl nitrate. The terms "ozone" or "peroxyacetyl nitrate" are used only when the method of measurement is specific.

2. Concentrations of Oxidants in Urban Atmospheres

a. Oxidant Concentration Patterns

Table 2-7 shows the maximum hourly average concentrations and peak concentrations, as well as the number and percentage of days when the maximum hourly average concentration exceeded 290, 200, and 100 $\mu g/m^3$ (0.15, 0.10, and 0.05 ppm) for 12 monitoring sites. These values vary greatly from city to city. The percentage of days that oxidant concentrations exceed 290 $\mu g/m^3$ (0.15 ppm) in Los Angeles and Pasadena are of an order of magnitude greater than in other cities. It is also interesting to note that peak concentrations in St. Louis reached 1670 $\mu g/m^3$ (0.85 ppm). The high peak concentration reported for the St. Louis air monitoring station, however, is one of a series of extraordinarily high readings that usually occur late at night and are of short duration. It is suspected that they result from emissions from a nearby large chemical complex rather than from atmospheric photochemical reactions. The cumulative frequency distribution of hourly average concentrations for the same 12 sites shown in Table 2-7 is presented in Table 2-8.

Table 2-7. SUMMARY OF MAXIMUM OXIDANT CONCENTRATIONS RECORDED IN SELECTED CITIES IN THE UNITED STATES, 1964-1967

Station	Total days of available data	Number and percent of total days with maximum hourly average equal to or greater than concentration specified						Maximum hourly average, ppm	Peak concentration, ppm
		0.15 ppm		0.10 ppm		0.05 ppm			
		Days	Percent of days	Days	Percent of days	Days	Percent of days		
Pasadena	728	299	41.1	401	55.1	546	75.0	0.46	0.67
Los Angeles	730	220	30.1	354	48.5	540	74.0	0.58	0.65
San Diego	623	35	5.6	130	20.9	440	70.6	0.38	0.46
Denver[a]	285	14	4.9	51	17.9	226	79.3	0.25	0.31
St. Louis	582	14	2.4	59	10.1	362	62.2	0.35	0.85
Philadelphia	556	13	2.3	60	10.9	233	41.9	0.21	0.25
Sacramento	711	16	2.3	104	14.6	443	62.3	0.26	0.45
Cincinnati	613	10	1.6	55	9.0	319	52.0	0.26	0.32
Santa Barbara	723	11	1.5	76	10.5	510	70.5	0.25	0.28
Washington, D.C.	577	7	1.2	65	11.3	313	54.2	0.21	0.24
San Francisco	647	6	0.9	29	4.5	185	28.6	0.18	0.22
Chicago	530	0	0	24	4.5	269	50.8	0.13	0.19

[a] 11 months of data beginning February, 1965.

Table 2-8. CUMULATIVE FREQUENCY DISTRIBUTION OF HOURLY AVERAGE OXIDANT CONCENTRATIONS IN SELECTED CITIES IN THE UNITED STATES, 1964-1965

City	Percent of hours with concentrations equal to or greater than stated concentrations, ppm								1964-1965 yearly average, ppm
	90	70	50	30	10	5	2	1	
Pasadena	0.01	0.01	0.02	0.04	0.12	0.18	0.23	0.26	0.042
Los Angeles	0.01	0.01	0.02	0.04	0.10	0.14	0.18	0.22	0.036
San Diego	0.01	0.02	0.03	0.04	0.08	0.10	0.12	0.14	0.036
Denver[a]	0.01	0.02	0.03	0.04	0.06	0.08	0.10	0.12	0.036
St. Louis	0.01	0.02	0.03	0.04	0.06	0.07	0.09	0.11	0.031
Philadelphia	0.01	0.02	0.02	0.03	0.06	0.08	0.11	0.14	0.026
Sacramento	0.01	0.01	0.02	0.04	0.06	0.08	0.10	0.12	0.030
Cincinnati	0.01	0.02	0.02	0.04	0.06	0.07	0.08	0.10	0.030
Santa Barbara	0.02	0.02	0.03	0.04	0.06	0.08	0.09	0.10	0.036
Washington, D.C.	0.01	0.01	0.02	0.03	0.06	0.07	0.09	0.10	0.029
San Francisco	0.01	0.01	0.02	0.03	0.04	0.05	0.06	0.07	0.019
Chicago	0.01	0.01	0.02	0.03	0.05	0.06	0.08	0.08	0.028

[a] 11 months of data beginning February 1965.

At most stations listed in Table 2-8, the concentrations are equal to or greater than 80 $\mu g/m^3$ (0.04 ppm) 30 percent of the time. Conversely, the concentrations are less than 80 $\mu g/m^3$ (0.04 ppm) 70 percent of the time. The differences observed among stations are almost entirely the result of the high rather than the low concentrations, even though the high concentrations occur less than about 10 percent of the time.

There appears to be no relationship between the ranking of stations by yearly average concentrations and the ranking by peak or maximum concentrations. The yearly average from Table 2-8 ranges from 37 to 82 $\mu g/m^3$ (0.019 to 0.042 ppm), which is approximately in the range of ozone concentrations found in remote, unpopulated areas.

Caution must be used, however, in the interpretation of this latter observation as well as of other conclusions that may be drawn from the data presented in Table 2-8. For example, one might, on the basis of yearly averages, conclude that Los Angeles, San Diego, Denver, and Santa Barbara had similar oxidant problems. Yet examination of the data in Table 2-7 shows that the peak concentration, maximum hourly average, and percentage of days with elevated oxidant concentrations are in fact quite different for these four cities. The principal reason for this apparent contradiction is associated with the nature of oxidant formation. Since ozone, the major oxidant, is a photochemical product and not a direct emission, the conditions necessary for its formation are restricted to the hours of sunlight. During any one day, therefore, the time when elevated oxidants can occur is restricted to a 4- to 6-hour period; at most, this time interval represents 25 percent of the 24-hour period. On this basis, 75 percent of the cumulative hourly data in Table 2-8 represents values that are close to zero. As a result, the differences that do exist between cities tend to disappear in the process of averaging. Thus, the usefulness and meaning of the yearly averages presented in Table 2-8 have serious limitations.

For similar reasons, the fact that yearly oxidant averages in urban areas approach atmospheric ozone background concentrations has little or no significance. The very rapid reaction between ozone and nitric oxide precludes the possibility of the presence of any nonurban-formed ozone in an urban atmosphere. Nitric oxide, unlike oxidant, is a direct emission and is emitted in substantial concentrations for most of the hours in a day; the yearly average in most cities is on the order of 50 $\mu g/m^3$ (0.04 ppm). If a background of 40 to 80 $\mu g/m^3$ (0.02 to 0.04 ppm) ozone is assumed, calculations indicate an ozone destruction rate of 1960 to 5880 $\mu g/m^3$ (1 to 3 ppm) per hour. Thus any nonurban-formed ozone would stand little chance of existing in an urban atmosphere. The yearly average oxidant values, therefore, represent oxidants formed photochemically in the urban atmosphere. These yearly values are low because 75 percent of the averaged values are necessarily near zero, as previously indicated.

Table 2-9 presents, on a yearly basis, the maximum hourly average concentration and the number of days when the maximum hourly

Table 2-9. SUMMARY OF TOTAL OXIDANT CONCENTRATIONS RECORDED AT CAMP SITES, 1964-1972 (24)

City	Year	Days of valid data	Number of days with at least 1 hourly average equal to or exceeding 0.05 ppm	0.10 ppm	0.15 ppm	Maximum hourly average, ppm
Chicago	1964	254	149	15	0	0.13
	1965	275	120	9	0	0.13
	1966	235	52	6	3	0.19
	1967	255	113	16	1	0.16
	1968	211	113	17	5	0.18
	1969	24	15	0	0	0.07
	1970	200	97	4	3	0.20
	1971	276	90	14	2	0.17
	1972	312	79	4	0	0.14
Cincinnati	1964	303	137	36	5	0.26
	1965	310	182	19	5	0.17
	1966	208	54	1	0	0.10
	1967	228	122	24	1	0.20
	1968	86	65	7	0	0.14
	1969	48	23	5	1	0.16
	1970	7	1	0	0	0.08
	1971	221	96	10	1	0.16
	1972	257	69	3	1	0.15
Denver	1965	285	226	51	14	0.25
	1966	298	187	46	9	0.19
	1967	166	76	12	4	0.21
	1968	151	149	28	5	0.26
	1969	108	70	2	0	0.13
	1970	141	69	9	1	0.18
	1971	184	63	5	1	0.20
	1972	209	94	26	1	0.18
Philadelphia	1964	269	124	37	9	0.20
	1965	266	109	23	4	0.33
	1966	315	145	52	19	0.52
	1967	282	124	28	3	0.17
	1968	140	88	18	3	0.21
	1969	92	37	3	0	0.11
	1970	112	29	10	0	0.13
	1971	260	47	5	0	0.14
	1972	47	20	10	1	0.15

Table 2-9 (Continued). SUMMARY OF TOTAL OXIDANT CONCENTRATIONS RECORDED AT CAMP SITES, 1964-1972 (24)

City		Days of valid data	Number of days with at least 1 hourly average equal to or exceeding			Maximum hourly average, ppm
			0.05 ppm	0.10 ppm	0.15 ppm	
St. Louis	1964	253	156	26	6	0.26
	1965	329	206	33	8	0.35
	1966	292	174	33	5	0.22
	1967	289	185	38	4	0.20
	1968	163	100	7	2	0.23
	1969	95	47	5	0	0.12
	1970	96	20	0	0	0.08
	1971	294	77	1	0	0.13
	1972	203	77	9	2	0.16
Washington,D.C.[a]	1964	293	163	40	4	0.20
	1965	284	150	25	3	0.21
	1966	325	134	27	2	0.16
	1967	322	137	27	5	0.26
	1968	217	134	40	9	0.25
	1969	71	30	1	0	0.10
	1970	167	95	10	2	0.16
	1971	299	144	17	0	0.13
	1972	179	45	6	0	0.13

[a] Site moved at end of 1968 to another section of the city.

average exceeded specified values at each of the CAMP* sites, from 1964 through 1972.

b. Seasonal and Diurnal Variations

Seasonal and diurnal variations in oxidant concentrations result largely from 1) variations in emissions of oxidant-forming pollutants, 2) variations in atmospheric transport and dilution of pollutants, and 3) variations in other atmospheric variables involved in the photochemical formation of oxidant. Typically, each of these factors varies significantly over periods as short as a few hours; the latter two also vary significantly seasonally. It is not unexpected, therefore, to find considerable variations in observed ambient oxidant concentrations.

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*CAMP - Continuous Air Monitoring Projects of the U. S. Environmental Protection Agency.

The highest monthly mean concentrations generally occur during the period from late spring to early fall. Oxidant concentrations exhibit daily as well as seasonal variations, and the maximum daily concentration generally occurs around the noon-hour, when the shorter wavelength solar radiation, which is photochemically important, reaches the surface of the earth with greatest intensity. Table 2-10 gives oxidant concentrations recorded in selected cities during the month having the highest mean 1-hour average concentration averaged

Table 2-10. HIGHEST MONTHLY MEAN OF 1-HOUR AVERAGE OXIDANT CONCENTRATIONS RECORDED IN SELECTED U. S. CITIES, 1964 AND 1965

Station	Month having highest mean 1-hour average oxidant concentration	Monthly mean of hourly average concentrations, ppm	Monthly mean of maximum daily 1-hour average concentrations, ppm
Pasadena	July	0.075	0.24
Los Angeles	August	0.056	0.17
San Diego	October	0.050	0.11
Denver[a]	July[a]	0.050	0.11
St. Louis	May	0.042	0.072
Philadelphia	July	0.054	0.11
Sacramento	June	0.040	0.075
Cincinnati	July	0.048	0.098
Santa Barbara	May and September[b]	0.042[b]	0.064 and 0.072
Washington	May	0.041	0.072
San Francisco	May	0.031	0.046
Chicago	April	0.044	0.070

[a] 11 months of data beginning February 1965.

[b] 1964-1965 average for the months of May and September.

for the years 1964 and 1965. For these months, the means of all hourly concentrations and the means of the maximum daily 1-hour average concentrations are also listed.

The seasonal variation of oxidant concentrations by month is illustrated for three of the sampling stations in Figures 2-4 and 2-5. Figure 2-4 illustrates the mean, by month, of all hourly average concentrations for Los Angeles, Denver, and Phoenix. Figure 2-5 shows the mean, by month, of daily maximum 1-hour average concentrations for the same cities. In these figures, the importance of solar radiation is readily apparent. Note that for Denver, the high values occur around mid-summer. For Los Angeles, the high values

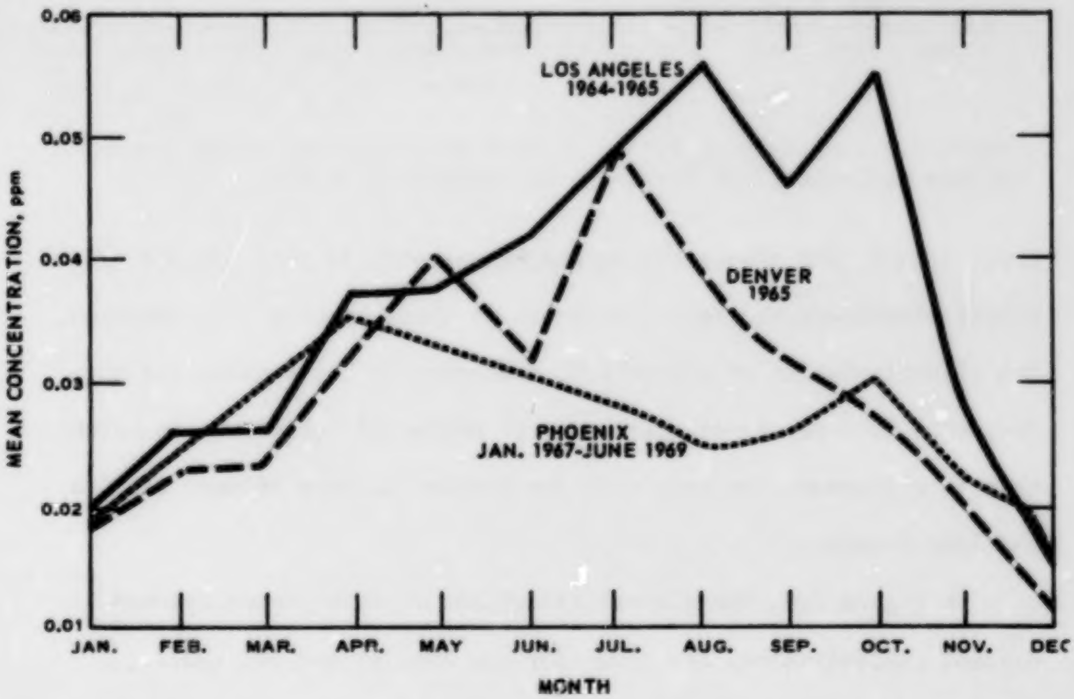

Figure 2-4. Monthly variation of mean hourly oxidant concentrations for three selected cities

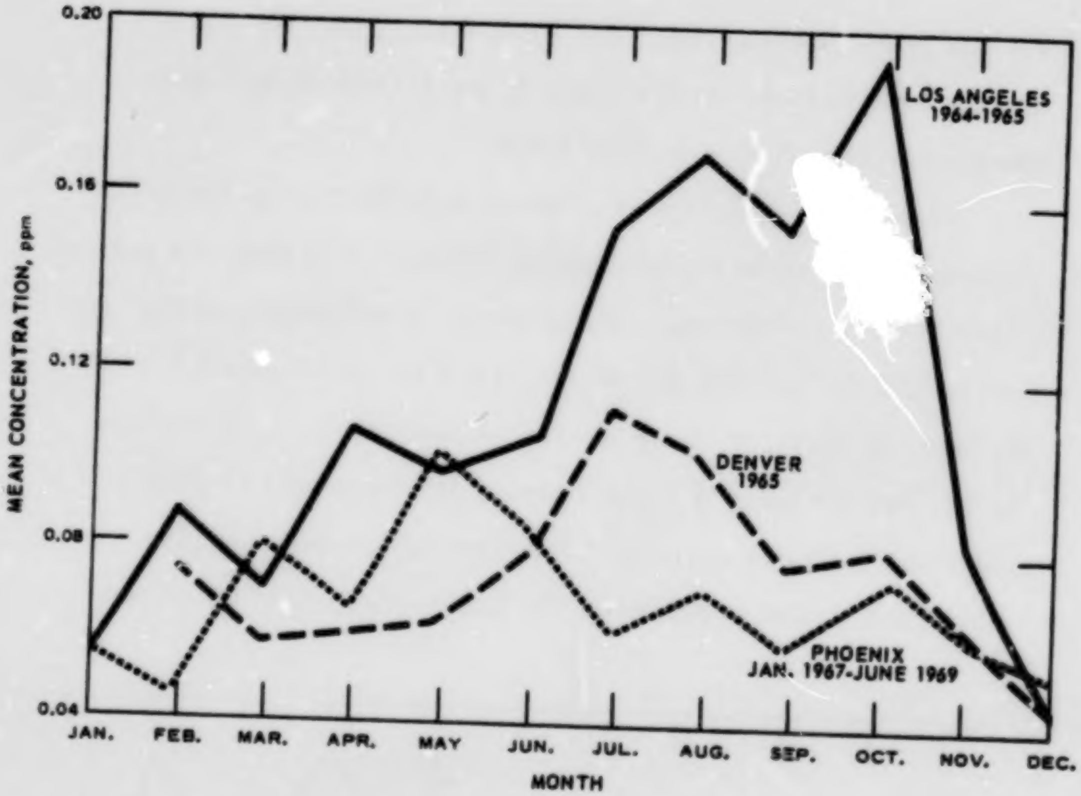

Figure 2-5. Monthly variation of mean daily maximum 1-hour average oxidant concentrations for three selected U. S. cities.

occur toward late summer and autumn, apparently in part because of slower windspeeds and less cloudiness in these seasons. In addition, the characteristics of atmospheric transport in Los Angeles are more favorable to a day-to-day carryover of precursor pollutants in autumn than in midsummer, the result of the greater balance between the sea and land breezes.

In Figure 2-6, the diurnal variations of mean 1-hour average oxidant concentrations are shown for Los Angeles and St. Louis. Selected for the Los Angeles presentation is the calendar month which had the highest monthly mean average for the years 1964 and 1965, while the St. Louis presentation illustrates the calendar month which had the highest monthly mean average in 1966. Figure 2-7 illustrates the diurnal variation of mean 1-hour average oxidant concentrations for a 3-day period, August 6-8, 1966, in Philadelphia when unusually high concentrations of oxidants were recorded. While there are some differences, all curves of Figures 2-6 and 2-7 show a distinct peak around noon. This peak results largely from the interaction of diurnal variations in emissions, solar radiation intensity, and atmospheric dilution.

Peak emissions occur with the morning rush-hour traffic, at a time when solar radiation and dilution are weak. As the emissions drop off toward midmorning and midday, radiation increases to a maximum around noon; dilution increases rapidly in the forenoon to

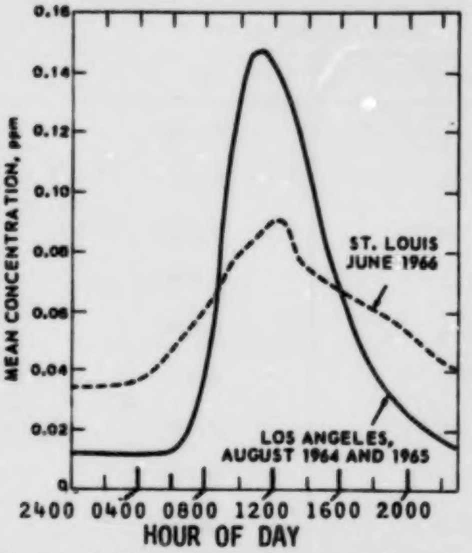

Figure 2-6. Diurnal variation of mean hourly average concentrations in Los Angeles, California, and St. Louis, Missouri.

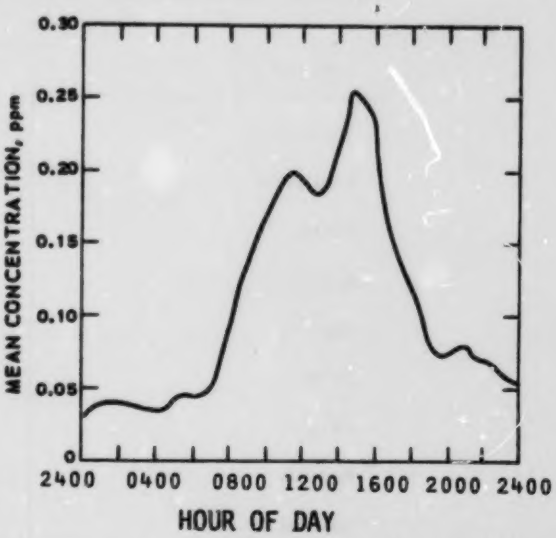

Figure 2-7. Diurnal variation of mean hourly average oxidant concentrations in Philadelphia, Pennsylvania, August 6-8, 1966.

reach a maximum around midafternoon. Consequently, the diurnal variations of oxidant concentration typically show a peak around noon.

c. Concentrations of Ozone in Urban Atmospheres

Tables 2-11 and 2-12 present the monthly average and mean of daily maximum ozone concentrations observed in Delft, The Netherlands in 1970 (16). The data from Table 2-11 and 2-12 are summarized in Figure 2-8. Table 2-13 presents a summary of the data for the days in which the maximum hourly ozone concentrations reached or exceeded 200 μg/m^3 in Delft during 1969, 1970, and 1971. Table 2-14 presents the ozone concentrations measured in 6 communities in the Netherlands beginning in May 1971.

Table 2-11. MEAN DAILY MAXIMUM OF HOURLY OZONE CONCENTRATIONS IN DELFT, 1970 (16)

Month	Ozone concentration	
	μg/m^3	ppm
January	4	0.002
February	23	0.012
March	90	0.046
April	104	0.053
May	116	0.059
June	121	0.062
July	88	0.045
August	140	0.071
September	113	0.058
October	58	0.030
November	46	0.023
December	29	0.015

Table 2-12. MONTHLY AVERAGES OF OZONE CONCENTRATIONS IN DELFT, 1970 (16)

Month	Ozone concentration	
	$\mu g/m^3$	ppm
January	2	0.001
February	13	0.007
March	50	0.026
April	58	0.030
May	48	0.024
June	45	0.023
July	46	0.023
August	61	0.031
September	50	0.026
October	32	0.016
November	25	0.013
December	10	0.005

[a] Figure 2-8 also.

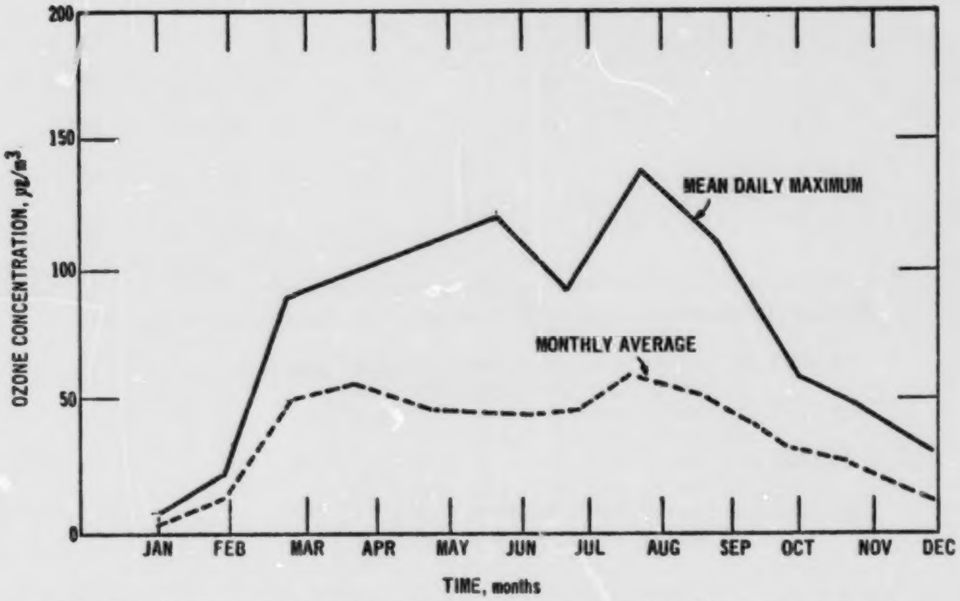

Figure 2-8. Monthly average of hourly ozone concentration and of daily ozone maxima for Delft, 1970.

Table 2-13. DAYS WHEN THE OZONE CONCENTRATION IN DELFT REACHED OR EXCEEDED A PEAK LEVEL OF 200 μg/m^3, 1969-1971 (16)

1969 day-mo	Maximum hourly average	1970 day-mo	Maximum hourly average	1971 day-mo	Maximum hourly average
9-4	310	10-3	190	15-4	248
5-5	395(440)[a]	2-5	190	4-5	200
15-6	306	14-5	287	6-5	191
16-6	285	20-6	218	11-5	214
1-7	205	21-6	313	31-5	275
16-7	370	30-7	198	1-7	259
23-7	196	1-8	190	5-7	192
4-8	191	13-8	230	8-7	208
9-9	192	29-8	217	9-7	226
13-9	204	30-8	227	11-7	239
		31-8	190	23-7	193
		18-9	237	30-7	249
		19-9	247	19-8	249
		20-9	237	21-8	245
		29-9	211	22-8	244
				29-9	186
				2-10	228
				3-10	364(>410)

[a] Values in parenthesis are the maximum peak concentration measured.

In 1969 340 hourly averages were >100 μg/m^3 and
22 hourly averages were >200 μg/m^3.
In 1970 550 hourly averages were >100 μg/m^3 and
30 hourly averages were >200 μg/m^3.
In 1971 845 hourly averages were >100 μg/m^3 and
48 hourly averages were >200 μg/m^3.
In 1972 236 hourly averages were >100 μg/m3 and
9 hourly averages were >200 μg/m^3.

Table 2-14. DAYS IN MAY THROUGH JULY 1971 WHEN THE OZONE CONCENTRATION AT ONE OR MORE SITES REACHED OR EXCEEDED THE HOURLY AVERAGE OF 200 μg/m^3 (16)

	Date, day-mo						
Monitoring site	27-5	31-5	1-7	8-7	9-7	11-7	30-7
Amsterdam C	169	213	119	163	214	215	--
Badhoevedorp	200	210	205	140	--	--	214
Naaldwijk	134	151	208	252	109	100	156
Delft	170	275	259	208	226	239	249
Vlaardingen	104	144	188	173	170	160	250
Hellevoetsluis	146	149	141	32	167	411	150

In the Federal Republic of Germany, the concentration of ozone has also been measured at two sampling sites, one in downtown Frankfurt/Main and the other at a nearby mountain station (Kleiner Feldberg, 800 m altitude). These investigations show that on days with intensive solar radiation, the ozone concentration at the downtown sampling site reaches as much as 100 ppb (Figure 2-9), whereas at the mountain site only about 40 ppb was measured (25). It is believed that the ozone in excess of background (50 ppb) at the downtown site results from photochemical reactions. Figure 2-10 shows average concentrations measured on all sunny days of the study period (25). The ozone concentration at downtown Frankfurt reaches, on the average, a maximum of about 40 to 50 ppb in the afternoon, while the simultaneous ozone concentration at the mountain station was 20 to 30 ppb.

The diurnal variation of mean hourly average ozone concentrations measured in 1966 and 1967 at two stations in the City of Berlin, Germany is shown in Figure 2-10a. The monthly averages from this measuring program is presented in Table 2-14a. The highest 30 minute average was 50 pphm ozone. The station in Steglitz was located in a street with high traffic density; Dahlem is a suburban station (44). It was felt that high levels of hydrocarbons at the urban station lead to lower oxidant levels.

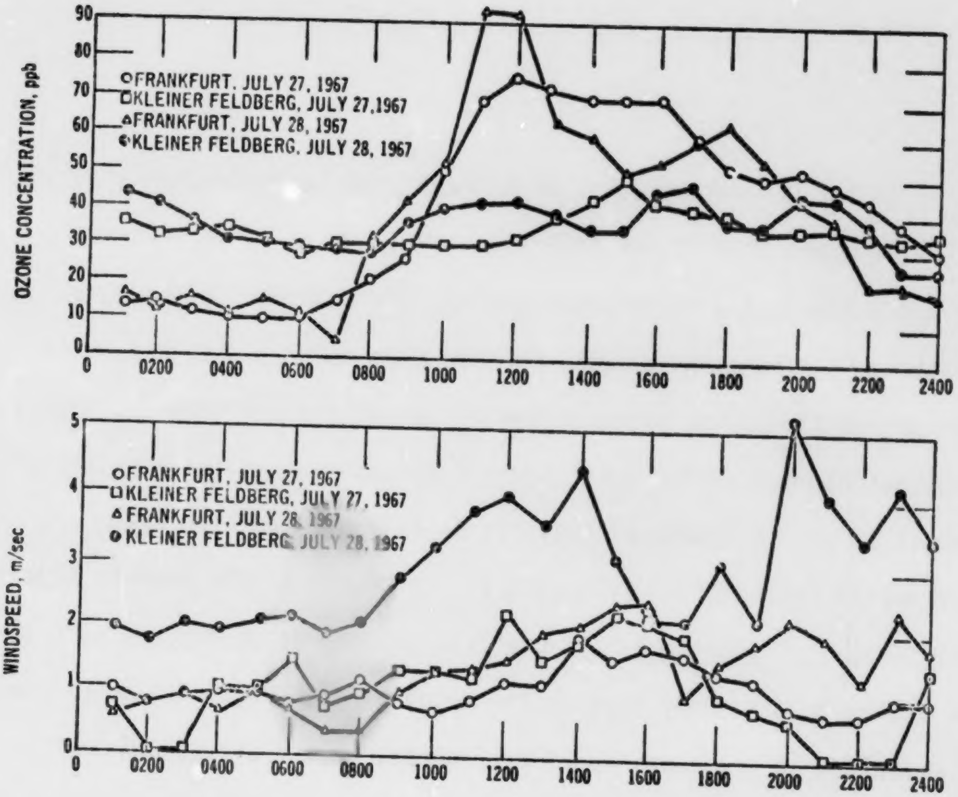

Figure 2-9. Diurnal variation of ozone at Frankfurt/Main and at Kleiner Feldberg mountain station on July 27 and 28, 1967.

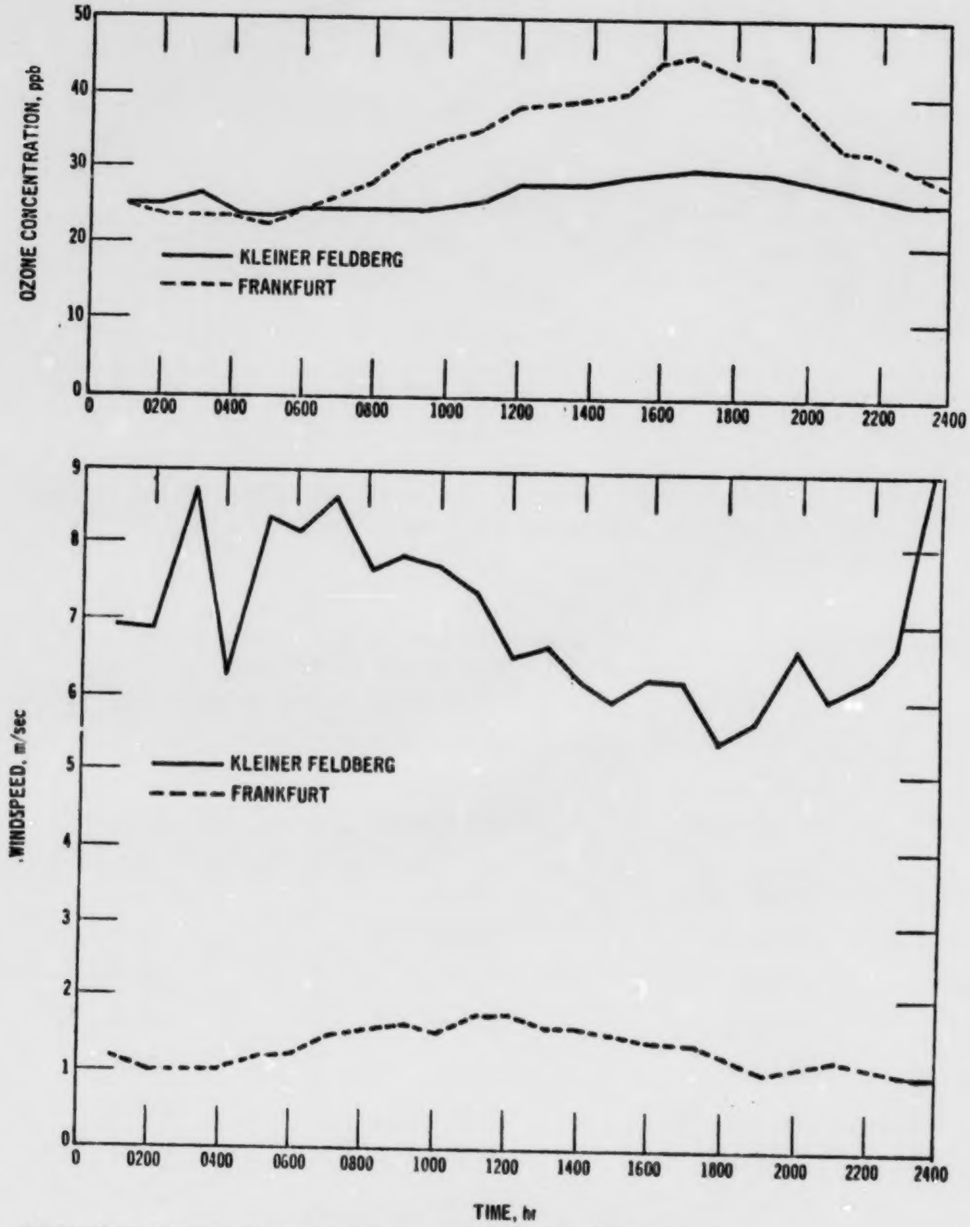

Figure 2-10. Concentration of ozone at a downtown Frankfurt/Main station and at Kleiner Feldberg mountain station on sunny days.

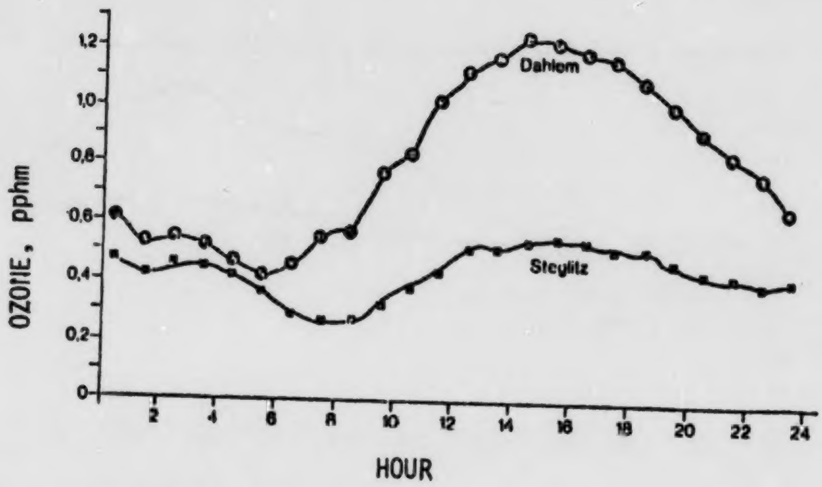

Figure 2-10a. Diurnal variation of ozone at two sites in Berlin, Germany, 1966-1967 (44).

Table 2-14a. MONTHLY AVERAGES OF OZONE CONCENTRATIONS AT TWO STATIONS IN BERLIN, GERMANY (44)

	Concentration, ppm	
Month--year	Steglitz (urban)	Dahlem (suburban)
August 1966	0.0043	0.0071
September	0.0045	0.0132
October	0.0119	0.0101
November	0.0014	0.0043
December	0.0017	0.0038
January 1967	0.0022	0.0039
February	0.0020	0.0026
March	0.0030	0.0037
April	0.0030	0.0040
May	0.0066	0.0107
June	0.0058	0.0118
July	0.0112	0.0230

d. Ozone Concentration Patterns

In one study made in the United States, the daily maximum 1-hour average ozone concentration was recorded for certain cities, and the number of days when this daily maximum exceeded 100,200, and 290 $\mu g/m^3$ (0.05, 0.10, and 0.15 ppm) is reported in Table 2-15 (26). Figure 2-11 shows the diurnal pattern of 1-hour average ozone concentrations measured on 2 selected days at the Denver and Philadelphia CAMP sites. This graph illustrates the high levels of ozone concentrations that have been measured in U. S. cities.

Figure 2-12 presents the diurnal variation of ozone concentrations measured in Amsterdam on March 10, 1971 (16).

Figure 2-13 presents a typical diurnal pattern of O_3, NO_2, NO, aldehydes, and hydrocarbons as measured in the University City of Rome (27). Figure 2-14 presents the ozone concentrations measured on selected days between February and May, 1972, in Ankara, Turkey (28).

Table 2-15. SUMMARY OF MAXIMUM DAILY 1-HOUR AVERAGE OZONE CONCENTRATIONS FOR SELECTED U. S. CITIES (26)

City	Time period	Number of days of valid data	Number of days with at least one hourly average equal to or exceeding			Maximum hourly average, ppm
			0.05 ppm	0.10 ppm	0.15 ppm	
Cincinnati	9/15/67 to 9/29/67	15	7	2	0	0.11
Washington, D.C.	10/3/67 to 10/16/67	15	3	0	0	0.07
Denver	10/17/67 to 11/1/67	15	7	1	1	0.19
	8/10/68 to 10/11/68	58	51	11	1	0.16
Los Angeles	11/2/67 to 11/19/67	18	16	14	5	0.26
Philadelphia	6/1/68 to 7/31/68	59	47	21	3	0.18

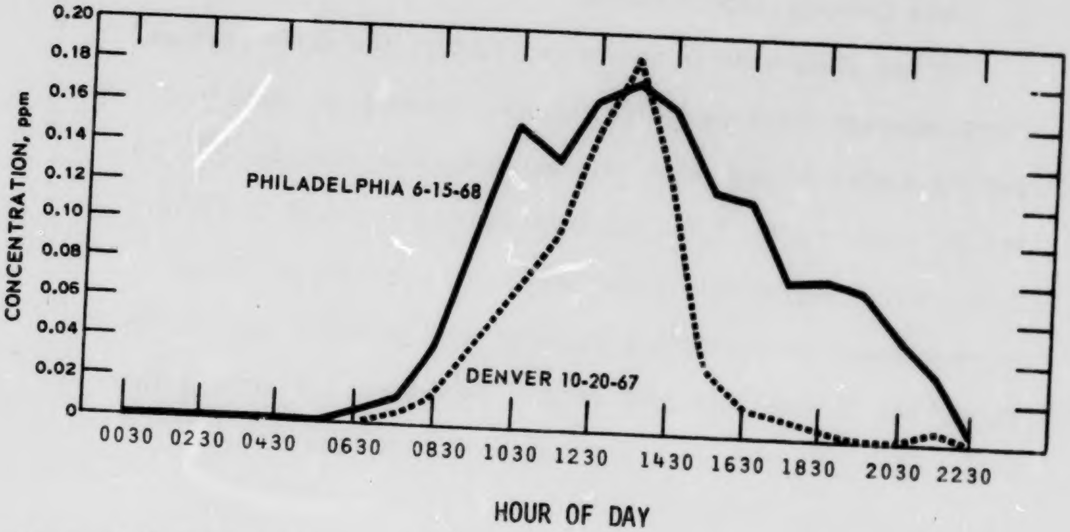

Figure 2-11. Diurnal variation of hourly ozone concentrations in Philadelphia and Denver.

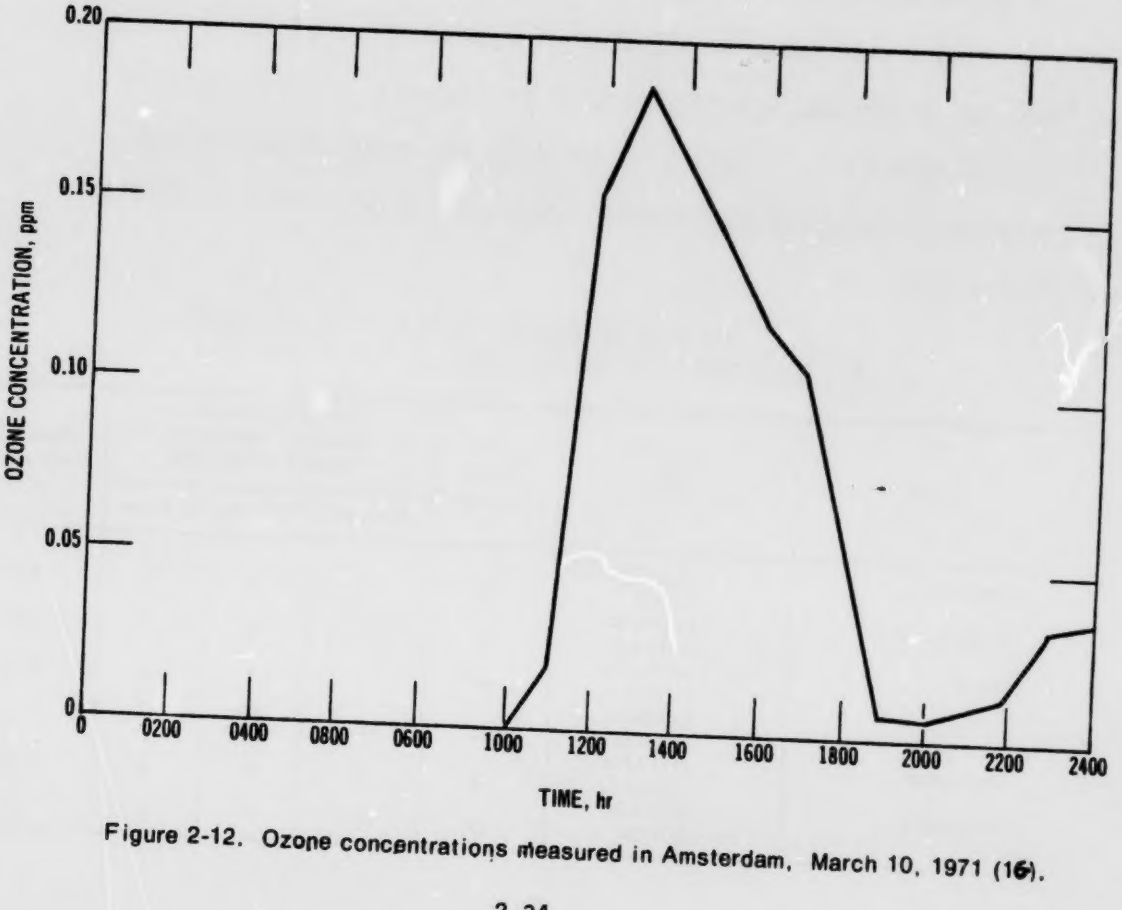

Figure 2-12. Ozone concentrations measured in Amsterdam, March 10, 1971 (16).

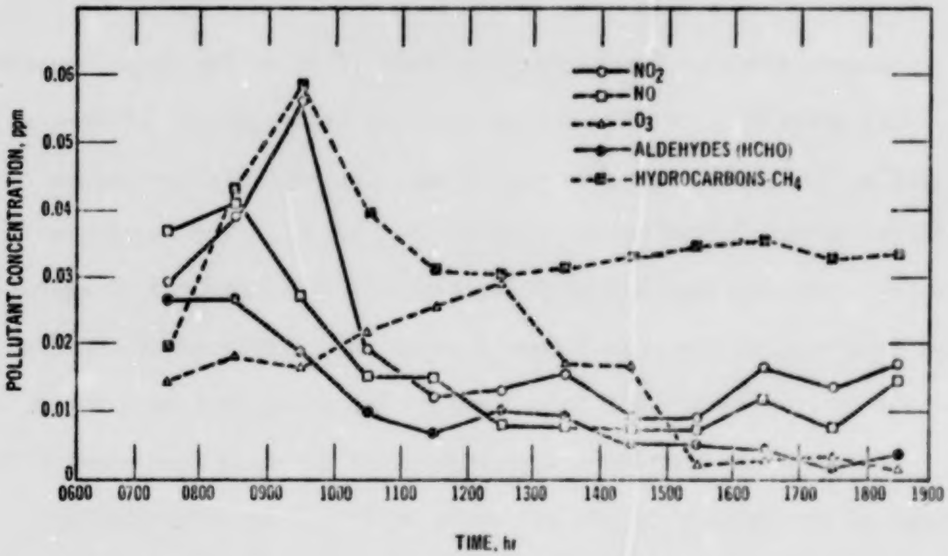

Figure 2-13. Determination of NO_2, NO, O_3, aldehyde, and hydrocarbon concentrations, University City in Rome.

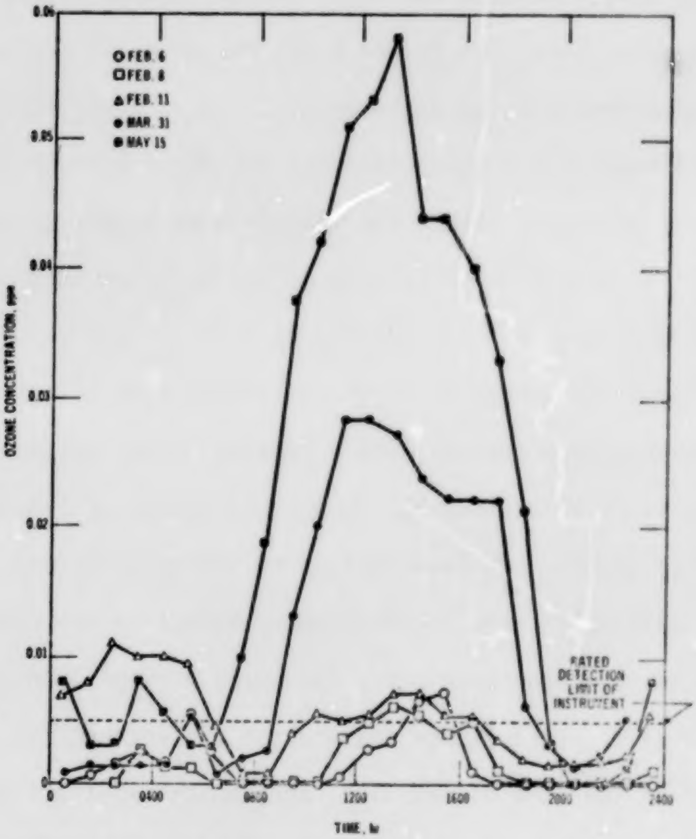

Figure 2-14. Diurnal variation in ozone concentration in Ankara, Turkey, on selected days of 1972 (28).

3. Concentrations of Peroxyacetyl Nitrate (PAN) in The Urban Atmosphere

Concentrations of PAN were measured in Los Angeles, California, utilizing gas chromatographic techniques with an electron-capture detector during September and October 1965 (29). Seven measurements per day were made for each of 16 weekdays in September and 19 weekdays in October. The mean 1-hour average concentrations of PAN and oxidant by hour of day for these periods are shown in Figure 2-15.

Beginning in June 1966, measurements of PAN have been made on the campus of the University of California at Riverside using the gas-chromatograph-with-electron-capture method mentioned. Samples are usually collected once each hour between 0700 and 1600 hr. Pacific standard time. In Figure 2-16, the mean 1-hour-average oxidant concentrations, as measured with a Mast analyzer, and the mean 1-hour-average PAN concentrations are shown by hour of day for the month of September 1966. The monthly mean hourly oxidant and PAN concentrations and the monthly mean of the daily maximum hourly average for 1 year are shown by month in Figure 2-17.

The comparison shown in Figure 2-17 is a good illustration of how specific averaging processes affect results. The considerable variations in daily maximum hourly concentrations as a function of time of year become much less obvious if the data include all hours. As stated previously, the latter damping effect is the result of including in the data those hours for which oxidant is necessarily at or near zero. Since these near-zero hours account for approximately 75 percent of the time of sampling, they have the effect of averaging out the elevated daytime values. Each method of averaging has its

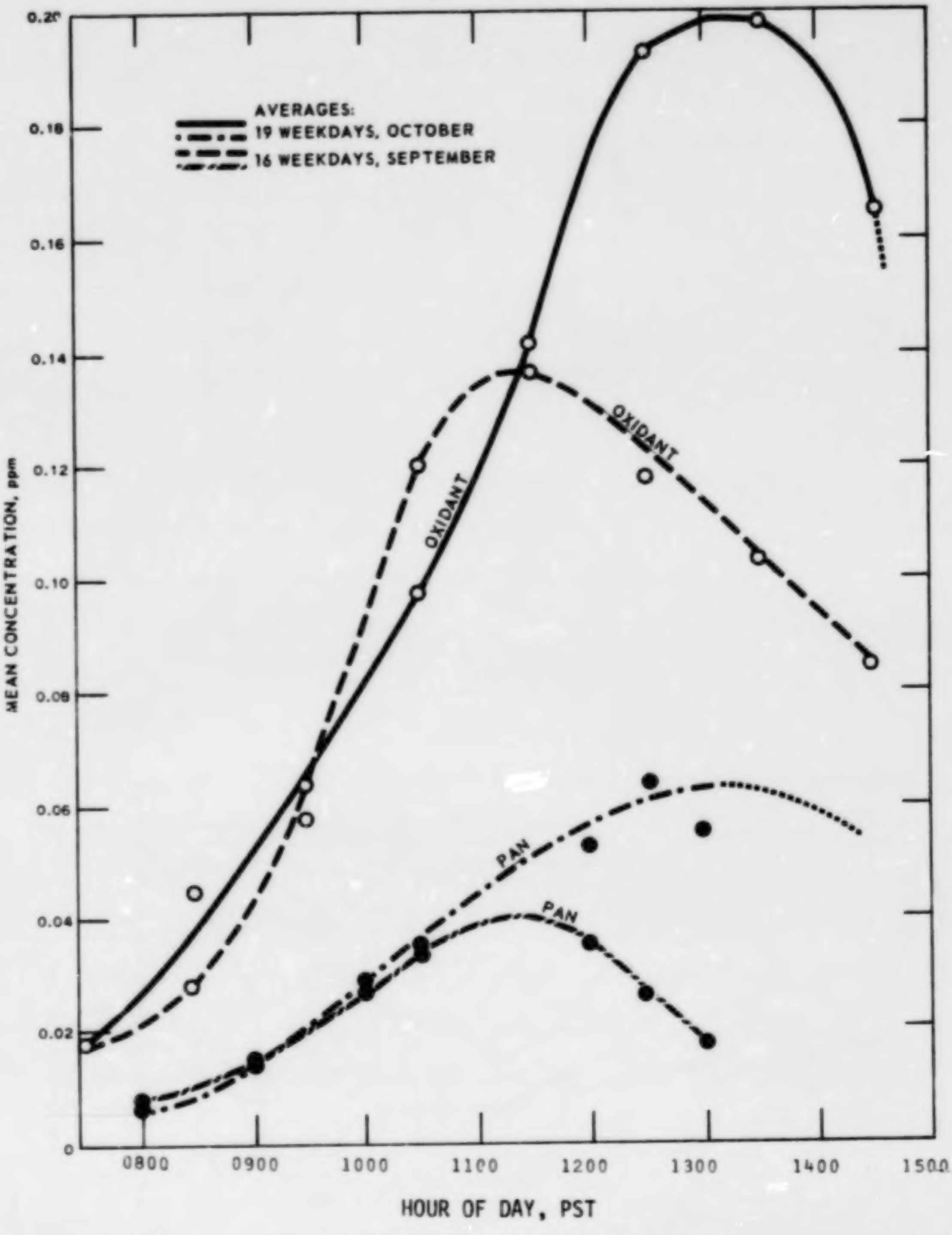

Figure 2-15. Variation of mean 1-hour average oxidant and PAN concentrations, by hour of day, in downtown Los Angeles, 1965.

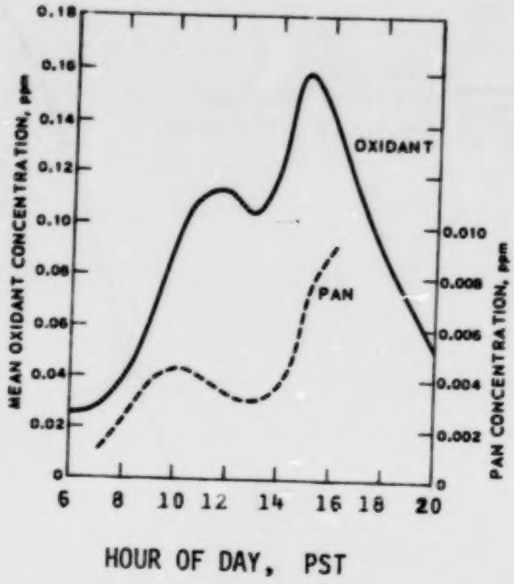

Figure 2-16. Variation of mean 1-hour average oxidant and PAN concentrations, by hour of day, at the Air Pollution Research Center, Riverside, California, September 1966.

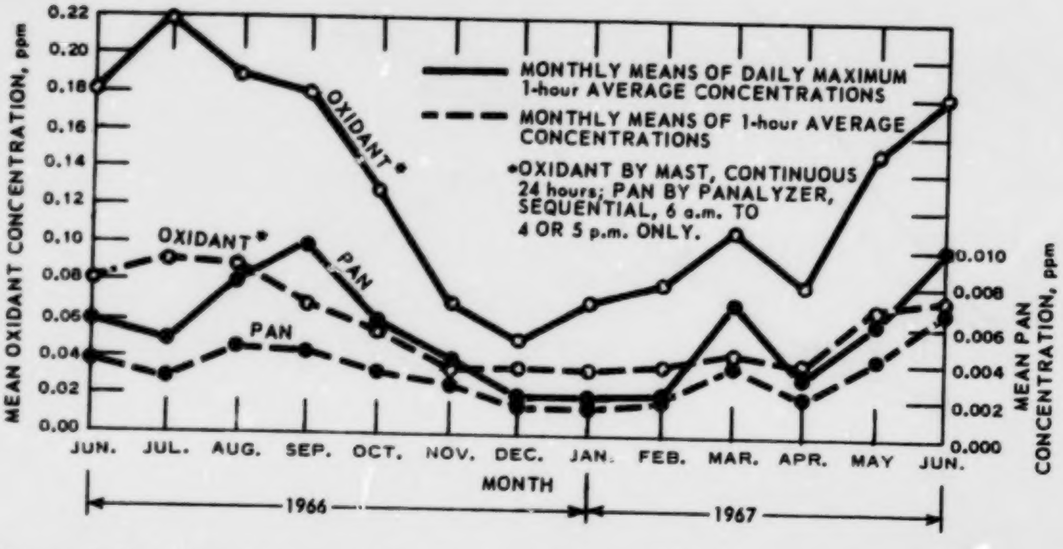

Figure 2-17. Monthly variation of oxidant and PAN concentrations at the Air Pollution Research Center, Riverside, California, June 1966- June 1967.

purpose, however, and subsequent interpretations of the results require careful consideration.

In Figure 2-16, there are two daily maxima for the oxidant and PAN concentrations. The PAN concentrations in Riverside are an order of magnitude lower than those in Los Angeles, while the concentrations of oxidants are of the same order of magnitude.

C. METEOROLOGICAL FACTORS

1. General Discussion

In any given area, the concentration of oxidant is dependent on many factors. Some of these, such as the concentration ratio of oxides of nitrogen to hydrocarbons, light intensity, and the reactivity of hydrocarbons, have been mentioned earlier. Other important factors are the size of the area, the meteorology, the topography, the number and distribution of sources, and the rates of emissions (30-32).

These latter factors are important because they affect the distribution of pollution over a city. Concentrations of oxidant on the upwind side of an area are substantially different from concentrations downwind. Oxidants may be transported many miles beyond the source area under certain meteorological and topographical conditions (32-34). These spatial variations in concentrations make the location of air monitoring stations of great importance if representative air pollution data are to be collected for an area or city.

2. Transport

The transport of pollutants by wind in the Los Angeles Basin has been the subject of several studies (32,35,36). Most of the trajectories enter the basin from the west and, in general, surface winds are predominantly from the ocean to the land during the spring, summer, and fall months.

To illustrate the eastward transport of oxidant, the diurnal variation of mean hourly average concentrations during October 1965, in West Los Angeles, Los Angeles, Azusa, and Riverside are shown in Figure 2-18 (37). The station at West Los Angeles is about 16 km west, and that at Azusa is about 32 km east, of downtown Los Angeles. The Riverside station is about 48 km east of Azusa. As shown, the time of the peak oxidant concentrations follows the time of peak concentrations in West Los Angeles by about 1 hour in Los Angeles, 2 hours in Azusa, and 4 hours in Riverside.

Oxidant concentrations at Riverside exhibited a double peak, as did the PAN and oxidant concentrations measured at the University of California at Riverside (Figure 2-16). The first peak at about 1100 hr is attributed to pollutants generated at or near Riverside, while the peak at 1600 hr is attributed to pollutants transported from the large and more densely populated Los Angeles metropolitan area.

As shown in Figure 2-19, which is a plot for carbon monoxide analogous to Figure 2-18, the afternoon peak concentration of carbon monoxide in Riverside is much smaller than the morning peak in Los Angeles. This suggests that the polluted air mass was diluted as it moved eastward to Riverside. On the other hand, the afternoon oxidant peak concentrations in Riverside were about as high as the peak con-

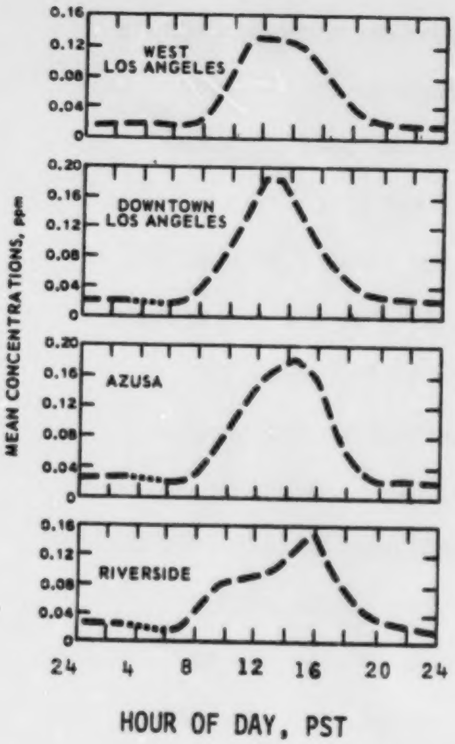

Figure 2-18. Diurnal variation of mean 1-hour average oxidant concentrations at selected California sites, October 1965.

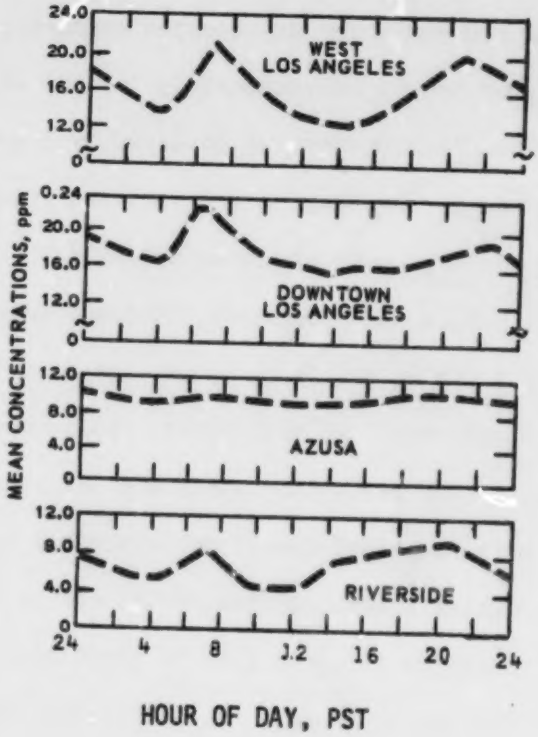

Figure 2-19. Diurnal variation of mean 1-hour average carbon monoxide concentrations at selected California sites, October 1965.

centrations in Los Angeles. It is possible, therefore, that, as the polluted air mass moved eastward, the oxidants continued to be formed at a rate about as great as the rate of dilution. It is also just as probable that the second peak at Riverside represents oxidant contributions from local as well as distant sources.

On the average, the direction of flow of surface wind and of pollutant transport appear to coincide, but they are not the only meteorological factors responsible for the transport of pollutants. Bell's (33) appraisal of the hour-by-hour development of oxidant concentrations indicates that other mechanisms, such as turbulence and downward motions created mechanically by airflow through mountain gaps, were also responsible for pollutant transport.

On some days, as observed by Stephens, the polluted air mass from the Los Angeles metropolitan area was defined by a sharp boundary that may not extend as far as Riverside (38). The reason for this sharp boundary, Stephens postulates, is that the temperature profile at the front of the air mass increased to the adiabatic lapse rate. At this point, rapid vertical ventilation had begun.

The prevailing winds are not always westerly. Under some meteorological conditions described by Bell (33), pollutants from Los Angeles have been transported out to sea and then southward to Oceanside (36) and even to San Diego, a distance of over 160 km. Under other conditions, pollutants have been transported from the sea northward to Ventura and Santa Barbara Counties (39,40).

Preliminary data from Santa Cruz, California, indicate that a similar phenomenon may occur in the region of the San Francisco - Oakland, California, metropolitan area. Under certain conditions, pollutants from the metropolitan area are transported out to sea and then brought back to shore by the local sea breeze to Santa Cruz, about 80 km south. On these occasions, the hourly average oxidant concentrations have been as high as 240 to 350 $\mu g/m^3$ (0.12 to 0.18 ppm) in Santa Cruz. The very low concentrations of oxides of nitrogen and hydrocarbons measured on these occasions again suggest substantial oxidant formation in spite of high dilution.

3. Monitoring Station Location

The Los Angeles County Air Pollution Control District has had extensive experience in measuring oxidants. Determination of the number of sites and selection of the locations was regarded by its designers as one of the most important factors in planning a continuing air quality survey (41). From the early 1950's until July 1955, 14 stations were maintained. The locations of the 14 stations were selected on the basis of surface wind trajectories, population, and industrial concentrations. On the average, each station represented an area of about 180 km^2.

After evaluation of the data obtained during 1957 and 1965, it became apparent that fewer permanent stations strategically located in the most frequent pattern of the smog masses could achieve adequate warning of hazardous pollution levels. Subsequently, the number of stations for this purpose was reduced to six.

The Bay Area Pollution Control District, San Francisco, California, also has a network of air monitoring stations. Examination of their data and data of the Los Angeles County Air Pollution Control District shows that average oxidant concentrations are not uniform throughout a metropolitan area.

In the Los Angeles Metropolitan Area during July 1965, for example, the mean of the daily maximum 1-hour average oxidant concentrations was 430 $\mu g/m^3$ (0.22 ppm) at Burbank, 470 $\mu g/m^3$ (0.24 ppm) at Pasadena, and 510 $\mu g/m^3$ (0.26 ppm) at Azusa. On the other hand, in the city of Los Angeles, which is southerly and adjacent to both the Burbank and Pasadena stations, this mean was 270 $\mu g/m^3$ (0.14 ppm). At North Long Beach, south of and adjacent to Los Angeles, it was 120 $\mu g/m^3$ (0.06 ppm).

Thus, the data from a single station may be indicative only of the concentrations in the immediate vicinity of that station. In order to determine local variations in air quality throughout an urban area, a complete air sampling network is necessary.

D. SUMMARY

Yearly averages of monthly maximum 1-hour average hydrocarbon concentrations, including methane, recorded continuously in various stations of the CAMP network, have reached 8 to 17 ppm (as carbon), but at least half of this amount is probably, in all cases the unreactive methane component. Thus, concentrations of 10 ppm (as carbon) or less of nonmethane hydrocarbons can give rise to observed concentrations of secondary photochemical contaminants in urban atmospheres.

The diurnal variation of hydrocarbon concentrations resembles that of carbon monoxide (at stations in the Los Angeles area) in having a pronounced maximum, which appears usually between 0600 and 0800 hr PST. It is not, however, parallel to the diurnal variation of secondary contaminants, particularly ozone, which typically show a pronounced maximum in the afternoon. This difference in behavior is characteristic of the photochemical air pollution complex.

For nonoxidant photochemical secondary contaminants, available information is limited to results of special studies on aldehydes in the Los Angeles area. These show that yearly maximum 1-hour average total aldehyde concentrations range from 0.20 to 1.30 ppm and that the analogous formaldehyde concentrations range from 60 to

150 μg/m^3 (0.05 to 0.12 ppm). Hourly average acrolein concentrations have been observed to range from 10 to 270 μg/m^3 (0.004 to 0.010 ppm) in various other studies. The ratio of formaldehyde to the total aldehyde index has been reported to be about 10 percent to about 90 percent. Much of the variation is undoubtedly caused by the use of different analytical methods in the different investigations.

Examination of the aerometric data from several major European, Turkish, and United States cities indicates the presence of photochemically formed oxidants. This is not an unexpected finding since the precursor reactants, hydrocarbons and oxides of nitrogen, are inevitable byproducts of current technology.

On a concentration basis, ozone has been identified as the major observed component of elevated oxidant levels. Demonstration of this fact, however, can be a most difficult task. One of the difficulties arises because some of the oxidant measuring methods in common use are nonspecific and subject to several interferences, although it is true that the major atmospheric components that cause these interferences have been identified and thus can be given appropriate consideration. Nevertheless, the inherent danger of oxidant concentration artifacts must be borne in mind.

A complicating feature of oxidants is the fact that their appearance in the atmosphere is dependent on chemical reactions, so that they occur, therefore, later in time than their precursor reactants. These chemical reactions are dependent on such variables as sunlight intensity, duration of sunlight, and temperature, in addition to variables affecting atmospheric dilution and dispersion. As a result, the relationship between precursor emissions and atmospheric oxidant concentrations is much less direct and more difficult to quantify than that relationship for primary pollutants.

The second class of photochemical oxidants, peroxyacyl nitrates, has not been routinely measured, although these compounds have been identified in the atmosphere of several cities. These hydrocarbon peroxy derivatives have been shown to be intimately related to atmospheric photochemical ozone formation, and therefore can be assumed to be present whenever oxidant levels are elevated.

Yearly averages are not representative of air quality with respect to oxidant pollution, however, because 1-hour-average ozone concentrations will necessarily be at or about zero for approximately 75 percent of the time, when photochemical reactions are minimal.

Air quality within a metropolitan region, on a statistical basis, shows gradients associated with prevailing patterns of wind movement. For primary pollutants, the greatest concentrations are to be found, on the average, in the near vicinity of the most prolific sources of emissions. For the secondary contaminants of the photochemical air pollution complex, the same generalization does not hold. Maximum concentrations of such photochemical products as aldehydes, nitrogen dioxide, peroxyacyl nitrates, and, especially, ozone are very likely to be displaced by 15 or 30 km from the areas in which the effective primary contaminants were emitted, and residual effects will be observed even farther downwind.

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CHAPTER 3

MEASUREMENT METHODS

A. INTRODUCTION

For most common gaseous air pollutants, the analyst must develop a specific analytical method for one or at most a few related species (1-3). Thus for many pollutants both simple manual and complex instrumental methods are available. This is not the case with hydrocarbons, for the complexity of the mixture of the compounds that may be present demands sophisticated instrumentation and techniques for measurement.

One instrumental technique can measure the total hydrocarbon concentration of the atmosphere. The value of the data obtained is severely limited, however, because the high background level of naturally occurring methane obscures the variation in levels of reactive hydrocarbon species, which are usually of principal interest because they may participate in photochemical reactions and methane does not. While simple attempts to eliminate the methane influence have had some success, only specific analysis provides the data needed to delineate properly and fully the nature of atmospheric photochemistry and to evaluate the relative contributions of various sources (4).

Total oxidants in the air may be defined as those compounds that will oxidize a reference material that is not capable of being oxidized by atmospheric oxygen. Methods for measuring oxidants, therefore, involve exposing an oxidizable substance to a sample of ambient air and determining the degree of oxidation that has occurred.

There are many such methods, each one being dependent upon the reference substance chosen.

Two widely used methods of determining the degree of oxidation are colorimetric and coulometric analysis of the iodine released by atmospheric oxidants from a potassium iodide (KI) solution. Since these methods are the most widely used, they will be discussed in detail.

It should be recalled that:

1. Ambient air contains a mixture of oxidizing and reducing compounds.
2. The concentrations of these compounds may be constantly varying within relatively wide limits-from 0 to 1 ppm.

Since reducing compounds in air have an opposite effect on the reference material--that is, cause a decrease in the degree of oxidation observed--the result obtained by these KI methods is a "net" oxidant value rather than a total value unless empirical corrections are made (5). Thus, the net value describes a condition of the air rather than a specific compound concentration. For this reason, concentrated efforts are being made by researchers to obtain measurements for each specific oxidant present. Hopefully these efforts will enable air pollution scientists to define better this atmospheric "condition."

Most oxidant data that have been collected are net determinations, and a high correlation between these values and other pollutant levels has been observed (6). Consequently, until the more promising methods become routinely available, net determination must serve as an indicator of total oxidant concentration.

It should be emphasized that in studies seeking a better evaluation of health effects, it is essential that data be obtained for individual oxidants, such as nitrogen dioxide, ozone, PAN, formaldehyde, acrolein, and organic peroxides, rather than for total oxidants. Instrumentation currently available permits the accurate measurement of atmospheric ozone, nitrogen dioxide, and PAN, but instrumentation must be developed that is capable of measuring the other individual gaseous pollutants that have the properties of oxidants.

Photochemical reactions and other problems resulting from oxidant pollution can be much better defined if specific methods are used to measure nitrogen dioxide and ozone in preference to the traditional methods used that determine just total oxidants.

B. CALIBRATION TECHNIQUES

Calibration at regular intervals is required for all analytical methods. It may be dynamic or static. Dynamic calibration involves passing known, realistic concentrations of the pollutant in air into the system as if it were an actual sample. This technique may be fairly difficult because of the very low concentrations of some pollutants and the reactivity or condensability of others.

A very efficient dynamic method is the exponential decay method (7). The required apparatus consists of a glass vessel containing a magnetically driven stirrer. The vessel is supplied with a carrier gas at a constant known rate; a known volume of test gas is introduced and, where mixing is complete, the gas concentration, C, decays with time according to the relationship $C = C_0 \exp - (Vt/U)$, where V is the volume of the vessel, U the gas flow rate, C_0 the initial gas concentration, and t the time elapsed after introducing the test gas.

Standard calibration gases in high-pressure steel cylinders are most commonly used to calibrate hydrocarbon analyzers. The gas is diluted in the cylinder to the desired concentration and then fed directly to the analyzer. Common hydrocarbons diluted to atmospheric concentration ranges are available commercially. Commercial mixtures should always be checked against a reference standard; or, if this is not possible, they should at least be checked by an independently calibrated hydrocarbon analyzer.

Various gas dilution systems have been described (8). A fairly simple method recently developed utilizes permeation tubes (9). This method is suitable for pollutants that can be condensed at moderate pressures in plastic tubes. The pollutant will effuse through the walls of a tube made of a specific plastic at a rate that is dependent upon tube surface area and temperature. To generate a known dynamic concentration, air is passed at a known rate over the permeation tube in a thermostated vessel. The tube may be calibrated gravimetrically if it is held at constant temperature, and the permeation rate remains constant as long as the tube contains appreciable liquid.

Another dynamic calibration technique involves the addition of a measured amount of pollutant to a known fixed volume of air in a large vessel. The addition may be made by syringe injection or by crushing a weighed glass ampoule containing the pollutant. If the vessel is rigid, it must be large relative to the amount of sample to be withdrawn. Bags made of plastics, which have the advantage of collapsing as the sample is withdrawn, may also be used. Bags must be made of an inert material to avoid changes in concentration through absorption or reaction with the walls of the bag.

Only dynamic calibration tests the integrity of intake lines, flow-measuring devices, filters, pumps, and other components of the analyzer that may contaminate or alter the sample before it reaches the sensor.

Difficulties in obtaining well defined ozone concentrations for calibration purposes led to the use of total oxidant methods for calibrating ozone methods. This should be borne in mind when considering the ozone results.

C. SAMPLE COLLECTION AND HANDLING

Samples for hydrocarbon measurement must occasionally be collected in the field and returned to a central laboratory for analysis. The manner in which they are transported must be chosen so as to prevent deterioration in storage and transit. Evacuated glass vessels or inert plastic bags are sometimes used for the collection of grab samples (10).

Alternatively, hydrocarbons and other organic compounds may be collected through a trap filled with a suitable adsorption material such as charcoal (11) or a gas chromatographic packing material (12).

When the former is used, the adsorbed compounds are extracted with a suitable solvent such as chloroform; when the latter medium is employed, desorption is accomplished by introducing an elution gas into a gas chromatographic column at fairly high temperature.

For condensable pollutants, a freeze-out technique may be used. The air is passed through a trap immersed in a cold bath (ice or liquid air) suitable for condensing the materials of interest. Sometimes the trap is packed with an inert solid adsorbent. Later the trap is connected to the detector and warmed to volatilize the sample. In this method, the normally large quantities of water vapor in air also condense. The water can sometimes be removed by a drying agent if the pollutant of interest is not affected as a result.

In most instances, grab samples can be analyzed by the techniques described above.

D. AEROSOL MEASUREMENTS

The particulate material observed in photochemical air pollution (in addition to that portion of solid particulates found even in the absence of a photochemical reaction system) is partially liquid in character. In a sample the moisture and other evaporable liquid generally present make weight and volume estimates difficult and dependent on the recent sample history. Prolonged passage of sample air over the aerosol may evaporate part of the collected material. The weight of a collected photochemical aerosol also depends on the relative humidity to which it has been exposed between time of collection and weighing (13). The extent to which gaseous photochemical reaction products condense to liquid and perhaps solid materials, or condense onto solid particulate material, is highly uncertain.

Thus aerosol collection, separation by particle size, and characterization are difficult. Most of the conventional collection methods are suitable for solid particles, but have highly uncertain effects on liquid aerosol. Methods based on light scattering do not affect the particles appreciably, but are hard to relate to known particulate sizes and densities.

E. METHODS FOR ANALYSIS OF TOTAL HYDROCARBONS

1. Flame Ionization

Originally developed as a detector for gas chromatography, the flame ionization technique (FID) was later adapted for total hydrocarbon analysis (14). In this technique, a sensitive electrometer detects the increase in ion intensity resulting from the introduction into a hydrogen flame of a sample of air containing any organic compound (e.g., hydrocarbons, aldehydes, alcohols). The response is approximately in proportion to the number of organically bound carbon atoms in the sample. The FID is essentially a carbon atom counter, but its response to carbon atoms in different compounds is nonlinear. As a result, FID data are usually expressed as the calibration gas used: for example, parts per million of carbon as methane. Carbon atoms bound to oxygen, nitrogen, or halogens give reduced or no response. There is no response to nitrogen, carbon monoxide, carbon dioxide, or water vapor;

but there is an oxygen effect, which can be minimized by appropriate operating conditions. The instrument also responds to hydrocarbon derivatives in approximate proportion to the carbon atoms bound to carbon or hydrogen.

The response of the FID is rapid and, with careful calibration, is sensitive to a fraction of a ppm carbon as methane. Variations in response to different hydrocarbons and derivatives must be accounted for in data evaluation (15). Practically all continuous hydrocarbon analyzers in use today utilize the flame ionization detector as the sensing element.

2. Spectrophotometric Methods

Spectrophotometric methods are usually applied to samples concentrated by freeze-out or other techniques. The principal problem encountered with their use is one of calibration. In many cases hexane is chosen as a calibration compound to represent the whole hydrocarbon class. In infrared spectrophotometric methods, the reading is made at about 3.4 micrometers, which is the carbon-hydrogen bond stretch wavelength. At this wavelength, more weight is given to saturated hydrocarbons, which are rich in carbon-hydrogen bonds, than to unsaturates. The absorbance at other infrared wavelengths can be read and correction made for certain principal components, such as methane, acetylene, and ethylene (1-3).

Nondispersive infrared spectrophotmetric instruments have similar limitations. Because instrument sensitivity is low, atmospheric analysis without freeze-out requires very long cell path lengths (1-3).

F. METHODS FOR ANALYSIS OF SPECIFIC HYDROCARBONS

1. Subtractive Columns

a. Nonmethane Hydrocarbons

In typical polluted air samples the principal hydrocarbon component, methane, is usually more abundant than all other hydrocarbons combined. Methane, however, does not participate in photochemical reactions. It is therefore desirable to measure methane separately (16). In one technique, a carbon column is treated with methane until it "breaks through," that is, until no more methane is absorbed even though other hydrocarbons are still retained (17). This column becomes a "methane only" analyzer when it is used to absorb nonmethane hydrocarbons before analysis of the sample by a flame ionization analyzer. If the methane analyzer is run in parallel or in alternation with a conventional total hydrocarbon analyzer, methane and total hydrocarbons are measured and, by difference, the nonmethane hydrocarbon concentration may be calculated. Thus, a better approximation of the reactive fraction is provided (18,19).

b. Reactive Hydrocarbons

Fractionation of organic compound contaminants can be useful when it results in separate determinations of classes of compounds having significantly different reactivities in the photochemical air pollution complex. Such a system, described by Klosterman and Sigsby (20), permits separate determination of three groups: 1) olefins and acetylenes, both aliphatic and alicyclic as well as, presumably, aromatic; 2) aromatics, excluding benzene; and 3) other hydrocarbons, including benzene. The members of the first group are scrubbed from a sample stream by a column containing firebrick supporting mercuric sulfate; those of the second group are removed by palladium sulfate. The first column also removes alcohols, ketones, and organic acids; the second is effective in scrubbing aldehydes, ethers, and esters. Though at present it is only used in the analysis of automobile exhausts, the system shows promise for use in conjunction with other hydrocarbon analyzers, including flame ionization and gas chromatographic units, in giving a complete definition of the nature of hydrocarbon pollution.

2. Gas Chromatography

Gas chromatography (GC) is the only available method for specific atmospheric hydrocarbon analysis (19,21). With flame ionization detection, it is sensitive in the part per billion range. Through the judicious choice of columns and temperatures, almost any desired separation of components can be effected.

Very few data are available on ambient levels of individual hydrocarbons, and none are available from the continuous monitoring of ambient air. Two factors have tended to produce this situation: GC data are obtained in a form such that it is quite tedious to reduce them to meaningful information; and, until recently, automation of data reduction was not possible and the manpower needed to process the volume of raw data manually was not available.

Qualitative identification of the many overlapping peaks of a gas chromatogram is difficult. An unknown can be identified, however, by comparing the chromatogram with that of reference compounds run under the same operating conditions, or by attaching a qualitative detector such as an infrared or mass spectrometer to the GC output. Both approaches require highly trained personnel.

For type separations using GC, subtractive columns may be used before or after the unit. Examples are mercuric perchlorate and silver sulfate-sulfuric acid columns, which are used to remove unsaturated hydrocarbons but pass paraffins (19). The use of subtractive columns simplifies an analysis in which individual components of different hydrocarbon types overlap or interface in the chromatogram.

At Battelle Institute, Frankfurt/Main, a simple and inexpensive mobile sampling unit for ambient air concentrations of hydrocarbons has been developed. The sample is drawn into small tubes filled with Porabek (silicon oil DC 200 Celite diatomaceous silica) at -196°C. The sampling time is a maximum of 1 hour and 10 to 100 liters of air must be sampled. The sample can be stored for some days at -2°C and for some hours at ambient temperature. The hydrocarbons are desorbed and analyzed by an advanced reversion gas chromatographic technique.

A method for the automatic analysis of C_1 - C_5 hydrocarbons in air has recently been published (22).

3. Spectrometric Methods

Both infrared and mass spectrometric methods are capable of considerable discrimination among hydrocarbons, but the low sensitivity required for ambient air monitoring often necessitates a freeze-out or concentration step. The operation and maintenance of either infrared or mass spectrometer is demanding and expensive, and data reduction is complicated. Infrared spectra may be used to determine the proportions of various olefins types, various aliphatic carbon-hydrogen types (primary, secondary or tertiary), and some aromatic types (23). The mass spectrometer can differentiate paraffin, olefin plus naphthene, and aromatic groups; in restricted narrow fractions it may permit analysis for individual components (24,25).

4. Methods for Olefins

A number of methods for olefin determination by colorimetric or coulometric techniques are available. The colorimetric reagents used include phosphomolybdate (26) and p-dimethylaminobenzaldehyde in sulfuric acid (27). A coulometric analyzer based on the reaction of olefins with bromine has been used successfully in several studies (28).

G. METHODS FOR ANALYSIS OF GASEOUS ALDEHYDES AND KETONES

1. General

Limitations of methodology allow the measurement of only a few specific oxygenated organics. Except for one formaldehyde procedure (29), there are no automated measurement methods for these contaminants. Only manual colorimetric procedures employing bubblers for sampling are available. The methods used for measurement of carbonyls (aldehydes and ketones) are mostly based on condensation reactions (30), and there is no single method that reliably measures total carbonyls or aldehydes. Sampling of oxygenates is difficult because of their water solubility and their ready condensation or adsorption in the analytical system.

2. Bisulfite Method

Heavier aldehydes are too insoluble for complex formation with bisulfite and most ketones form unstable complexes. The bisulfite aqueous reagent (31) forms moderately stable complexes, however, with lower-molecular-weight aldehydes and methyl ketones (32). After complex formation, the excess reagent is destroyed and the complex is broken up and the released bisulfite is analyzed by iodimetry. This method is only moderately sensitive. Bisulfite reagent is also used as a collection absorber for other methods because the carbonyls are readily liberated by acidification of the complex.

3. Other Condensation Reagents

Among the condensation reagents that will react with carbonyl compounds (1-3) are: cyanide, hydroxylamine, phenylhydrazine, 2,4-dinitrophenylhydrazine, Schiff's reagent (29,69,70), 2-hydrazinobenzothiazole, 3-methyl-2-benzothiazole hydrazone (MBTH), and chromotropic acid (33). The Schiff's reagent and chromotropic acid methods can result in good precision and sensitivity for formaldehyde measurement when used with current analytical procedures. The MBTH method is good for aliphatic aldehydes (including formaldehydes), but purity of the MBTH is a constant problem. Since it reacts with both aldehydes and ketones (34-36), 2,4-dinitrophenylhydrazine is one of the more general carbonyl reagents used (37). Colorimetric determinations with 2,4-dinitrophenlhydrazine are complicated by shifts in wavelength with type of carbonyl compound; gravimetric analyses with 2,4-dinitrophenylhydrazine are lengthy and too insensitive for atmospheric work.

Formaldehyde, generally found to be the predominant carbonyl compound in the atmosphere, is very water-soluble and in most derivatives is not typical of aldehydes as a class. For this reason, it is often desirable to use a procedure specific for formaldehyde (such as the chromotropic acid method), (33,38) or the method based on Schiff's reagent (29,69,70), and then determine total aldehydes or carbonyls by another method, subtracting the amount of formaldehyde found independently.

The unsaturated aldehyde, acrolein, has also received special attention because, like formaldehyde, it is a known lacrimator found in measurable concentrations in photochemically polluted air. The method used most commonly to measure acrolein is colorimetric and employs a 4-hexylresorcinol reagent, which is specific and sensitive (39).

H. METHODS FOR MEASUREMENT OF TOTAL OXIDANTS

1. Continuous Methods Utilizing Potassium Iodide

a. Colorimetric

Continuous analysis of total oxidants is most commonly accomplished with instrumentation, using a 10 to 20 percent solution of neutral-buffered potassium iodide as the absorbing medium. In measurement devices based on colorimetric principles, sample air is passed at a known rate through a liquid absorbing reagent with a vacuum-pump arrangement. Oxidants in contact with the KI react to free iodine and the triodine ion. These are determined colorimetrically in a continuous flow colorimeter at 3520 Å . The continuous instrumental method is extremely sensitive to oxidizing substances and is suitable for determinations of 0 to 1000 $\mu g/m^3$ (0 to 0.5 ppm) ozone. The negative interference caused by sulfur dioxide is often reduced by passing the air sample over chromium trioxide (CrO_3) scrubbers, but this technique has not been entirely successful, primarily because the effects of humidity on scrubber systems often render them ineffective (40). These scrubber systems also convert a portion of the ambient NO to NO_2, which thus necessitates an additional correction factor. Despite these drawbacks, CrO_3 scrubbers remain the most generally used technique for removing SO_2 interferences; they are particularly useful for this purpose during periods when ambient air contains high SO_2 concentrations. Nevertheless, better methods for eliminating SO_2 interferences are critically needed.

b. Coulometric

A coulometric method is also widely used for measurement of oxidants. In devices using this technique, sample air is passed, similarly as in the colorimetric type of instrument, into an electrolytic-detector cell containing KI (41). The free iodine liberated by the oxidants is reduced at the cathode of the cell, causing a current flow through an external circuit. The current flow is proportional to the amount of iodine liberated, and, in turn, to the oxidants entering the solutions. The current is measured with a microammeter that is usually calibrated directly in parts per hundred million (pphm) of ozone. The method can be made much more selective for ozone by using a differential system (42), which is described in Section I.2.

c. Colorimetric versus Coulometric Methods

Since colorimetric and coulometric instruments are the most widely used for oxidant measurements, there have been studies conducted that compare the results obtained with these respective methods. Field comparisons of measurements from colorimetric and coulometric oxidant analyzers, where readings did not exceed 390 $\mu g/m^3$ (0.20 ppm) and corrections were made for NO_2 interferences, indicate that differences are no greater than would be expected if two colorimetric instruments were working side by side (43). Comparative data, including readings greater than 390 $\mu g/m^3$ (0.20 ppm), show a correlation coefficient of 0.87 between readings of the two different instruments, both calibrated with known ozone-concentrations (44). In spite of the relatively good correlation obtained in this study, there were some instances when the agreement between the two methods was poor. To resolve some of these discrepancies, more comparative studies of this type are needed.

Perhaps the largest variations between the two methods are related to the differences in reagent formulation. Generally, 10 to 20 percent KI solutions are used in colorimetric instruments; at these concentrations, response to nitrogen dioxide is higher than at the 2 percent level used with coulometric instruments. Results of several studies are shown in Table 3-1. Analyzers utilizing other principles of detection vary in their sensitivity to nitrogen dioxide. As shown in Table 3-1, the coulometric cell analyzers register about 10 percent of the nitrogen dioxide concentration as ozone, and the U. V. photometric device shows the least interference from nitrogen dioxide.

Studies comparing the two methods indicate that both instruments must be checked and calibrated frequently and that spurious variations in readings are usually caused by changes occurring in the airflow or reagent flow and by interfering compounds. Table 3-2 gives some

Table 3-1. EFFECT OF NITROGEN DIOXIDE ON OXIDANT DETERMINATION IN THE ABSENCE OF OZONE

Nitrogen dioxide concentration in air,[a] ppm	Method	NO_2 concentration (as ozone), %	Reference
>0 to 36	1% KI, pH 7 bubbler	8 to 11	45
1.5 to 5.5	2% KI, pH 7 bubbler	6.4	46
2.0	10% KI, pH 7 contact column	21	44
>0 to 1	20% KI, pH 7 contact column	30	45
0.24 to 0.43	20% KI, pH 7	12 to 47 (average= 25)	47
2.0	coulombic cell	10	44
2.0	U. V. photometer	2	44

[a] Based on 4-liter sample.

Table 3-2. CALIBRATION STABILITY OF COLORIMETRIC OXIDANT MONITORING STATIONS, STATEWIDE CONTINUOUS AIR-MONITORING NETWORK, CALIFORNIA, 1963 to 1965

Change from previous calibration, %	Number of stations	Occurrences	
		Number	% of total
± 0 to 10	7	20	62.3
±11 to 20	4	7	22.0
±21 to 30	2	3	9.4
±31 to 40	2	2	6.3

indication of the reliability of the continuous colorimetric oxidant analyzers used at seven California stations. These analyzers are dynamically and statically calibrated about twice a year. The percentage of change from the previous calibration indicates the uncertainty in measurements obtained during the time between calibrations. Table 3-2 shows that 62 percent of the time there was less than a 10 percent change, and 78 percent of the time there was less than a 20 percent change. Changes greater than 20 percent occurred at three of the seven stations, which indicates that some stations provide more reliable data than others.

2. Other Methods - Intermittent Sampling

a. Ferrous Ammonium Sulfate

This method is based on the conversion of the ferrous ion by oxidation to the ferric ion. Samples of ambient air are drawn through an acidified ferrous ammonium sulfate solution. The ferric ion formed in the collection solution is complexed in the laboratory with ammonium thiocyanate, and the resultant color is measured on a spectrophotometer. The oxidized solution is stable and can be analyzed later. Depending on the sample flow rate and interval of collection, this method is useful in the range of 1 to 2400 $\mu g/m^3$ (0.0005 to 1.2 ppm) oxidant. As with the KI method, this method suffers from the drawback of interferences from reducing compounds in the atmosphere. Another weakness is the lack of agreement with results obtained by other methods.

b. Alkaline Potassium Iodide

This method (40) is intended for the manual determination of oxidant concentrations range from a few $\mu g/m^3$ to about 39,000 $\mu g/m^3$

(20 ppm). The advantage of this procedure over the neutral iodide technique is that a delay between sampling and completion of analysis is allowed. Sampling is conducted in midget impingers containing 1 percent KI in a 1-normal sodium hydroxide solution. Because of the simplicity of the neutral-buffered KI method, however, the alkaline procedure is not widely used.

c. Phenolphthalin

The phenolphthalin method is based on the oxidation of phenolphthalin to phenolphthalein by the oxidant present in the atmosphere. Because the technique responds to pH and temperature changes, it is not widely used at present, although it was used extensively to obtain oxidant data in the past. Measurement results obtained by this method are approximately twice those obtained by the KI method. Consequently, when data obtained by the two methods are compared, it is necessary to adjust for this difference.

I. SPECIFIC METHODS FOR OZONE MEASUREMENT

1. Chemiluminescence

Three different chemiluminescence techniques have been applied to the measurement of atmospheric ozone (48). Regener has reported on a surface ozone monitor that uses the principle of chemiluminescence (49,50). In Regener's approach, chemiluminescence is obtained upon reaction of ozone at a surface of Rhodamine-B dye adsorbed on activated silica gel. The intensity of the chemiluminescence, which is measured with a photomultiplier tube, is a linear function of ozone concentration passing over the surface. This technique is sensitive to less than 0.001 ppm O_3, and no atmospheric interferences have been observed (51). Prototype Regener monitors have shown excellent performance in extended field evaluations (52). The construction of the automated monitor, however, is complicated by the necessity of incorporating frequent internal calibration cycles to compensate for changes in the sensitivity of the chemiluminescence surface. The method can be made more stable by protecting the dye with gallic acid and using an intermittent sampling procedure (53).

The gas-solid chemiluminescence approach has been largely supplanted by gas-phase chemiluminescence techniques that have better response characteristics (54). This technique is based on a chemiluminescent reaction, at atmospheric pressure, between ozone and ethylene (55,56). Air (1 liter/min) and ethylene (10 to 20 cm^3/min) mix near the cathode of a photomultiplier. The chemiluminescence from the ozone-ethylene reaction is in the 4300 Å region (54). No atmospheric interferences have been observed and no filter is needed between the reaction cell and the photocathode. The chemiluminescence intensity is a linear function of ozone concentration and the sensitivity is about 0 001 ppm. Prototype monitors of this type have been constructed. Commercial versions of the Nederbragt detector, as it is called, are now being offered by several manufacturers. The gas-phase technique is used by the U. S. Environmental Protection Agency for corrected oxidant measurements (57).

Fontijn et al. (58) have developed a gas-phase technique that utilizes the chemiluminescent reaction between nitric oxide and ozone at low pressures. If one of the components is in excess, the chemiluminescence intensity obtained is proportional to the concentration of the second reactant. The air sample is mixed with reactant gas inside a low pressure (1 to 5 Torr) reaction chamber that is closely coupled to the photocathode of an infrared-sensitive photomultiplier. Sensitivity is 0.005 ppm for detection of either NO or O_3, and no atmospheric interferences are observed when the appropriate light filter is used. One advantage of this sensor over the other chemiluminescence types is that the same instrument may be used to measure NO or O_3. A disadvantage is the requirement for low-pressure operation. This detector has been used rarely for ozone measurements, but it is being used extensively for the measurement of oxides of nitrogen.

2. Differential Galvanic Method

An automatic atmospheric ozone analyzer has been developed that can perform measurements over periods of several weeks (42). The principle used involves the liberation of halogen by ozone from a phosphate-buffered solution of halide; then the halogen concentration is determined galvanometrically. A differential measuring technique by means of a galvanic cell with two cathodes and one counter electrode is used; background and noise current of the detector are extremely low and stable. The noise is equivalent to about 0.6 $\mu g/m^3$ of ozone, and the noncumulative drift over a period of several weeks is equivalent to not more than 6 $\mu g/m^3$ of ozone. The time constant is about 40 seconds.

Selectivity of the instrument for ozone has been demonstrated by laboratory experiments and in field testing and, based on an ozone-to-halogen stoichiometry of 1:1, the instrumental results obtained in field testing are in excellent agreement with those obtained by other techniques applied to and described for the determination of ozone.

3. Ultraviolet Photometry

Specific determination of atmospheric ozone may be accomplished by use of u.v. absorption measurements at 2537 Å (48,59,60). Interferences may be eliminated in a double-beam instrument by passing the air to the reference cell through a tube, coated on the inside walls with potassium permanganate, that selectively removes ozone. This is perhaps the most direct way of monitoring ozone in the atmosphere. The advantage of this method over chemiluminescence is that the measurement for ozone can be absolute since the absorption coefficient is well known. The Kruger ozone photometer was available in the past, but because of electrical and thermal instability, it was not extensively used. A much-improved instrument which uses the ultraviolet photometry has recently been marketed, and has been described by Behl (61).

4. *Trans*-butene-2 Gas-Phase Titration

This method involves a periodic gas-phase titration of the air sample with *trans*-butene-2 prior to analysis for total oxidants (62). This reactive olefin selectively removes the ozone present in the air stream and provides a specific ozone concentration by difference. The technique is applicable to coulometric and colorimetric methods of iodine measurement but has been evaluated more fully with a colorimetric instrument (63).

5. Other Chemical Methods

Hauser and Bradley (64) have described a specific ozone technique using 1,2-di-(4 pyridyl) ethylene as the absorbing medium and colorimetric analysis of the resulting aldehyde by the 3-methyl-2-benzothiazolone hydrazone method (40). This method can be used in the field analysis of atmospheric ozone because sampling and analysis need not be performed on the same day.

Hydrogen peroxide, 1-hexene, and PAN were found to interfere with this technique when a 24-hour sampling period was used. Glacial acetic acid, which is used in the collection solution, also presents a problem since it cannot be sent through the mail. Furthermore, the absorbing solution that is used freezes at 16° C and must be artifically warmed if used below this temperature. For a 30-minute sample collected in a bubbler at 0.5 liter/minute, the range of measurement for this technique is 100 to 2300 $\mu g/m^3$ (0.05 to 1.2 ppm) ozone.

A simple colorimetric method was described by Dorta-Schaeppi and Treadwell (65), who used the bleaching of indigo sulfonic acid by ozone. This method proved to be selective and to correlate well with other methods (66).

J. METHODS FOR DETERMINATION OF PEROXYACETYL NITRATE

1. Long-path Infrared Spectroscopy

Peroxyacyl nitrates were discovered by means of long-path infrared spectroscopy. Although water vapor interferes with the principal absorption band used in this determination, an alternate band may be used to avoid this interference. Strong bands are located at 5750 and 8620 nm and weaker bands at 7300, 9500, 10,000, 10,800, and 12,200 nm. A 500-meter long-path infrared cell is used to detect PAN in polluted ambient air (67).

2. Gas Chromatography

Increased sensitivity for the detection of PAN is provided by gas chromatography using an electron-capture detector (68). The advantage of this detector is its relative insensitivity to other compounds that might interfere.

Short columns (230 to 460 mm in length by 3.2 mm in diameter) packed with carbowax coated on an inert substance are used for the separation. Retention time for PAN is 1 to 2 minutes. Two-milliliter samples containing 5 $\mu g/m^3$ (0.001 ppm) PAN give detectable peaks. This method has been automated, but currently it is used primarily as part of a manual sampling program.

K. SUMMARY

With a few exceptions, atmospheric hydrocarbon measurements are made with continuous-monitoring, relatively complex instruments. Increasing demands for detailed data on specific compounds will reinforce this trend.

Continuous-analysis instrumentation demands dynamic calibration techniques. Standard gases for this purpose are commercially available, may be generated by permeation tubes or dilution systems, or may be prepared in large containers.

Flame ionization analyzers are sensitive, reliable, and suitable for the continuous measurement of total hydrocarbons. They fail, however, to give the specific detailed information required for a thorough understanding of the atmospheric photochemical problem. Attempts to further define the hydrocarbon mixture by using pretreatment columns to measure only methane or various reactive classes have met with some success in limited application.

Spectrometric techniques both for total and specific analyses are complex and generally insensitive; they require sample concentration steps or long cell-path lengths.

Gas chromatographic analysis provides the requisite sensitivity and specificity for the quantitation of individual hydrocarbons. Difficulties in qualitative analysis and data handling have limited the application of GC to short-term studies for the most part, and no continuous data are available.

Carbonyl compounds, specifically aldehydes and ketones, can be measured by several manual colorimetric techniques, but few actual data are available.

The most widely used method for the analysis of atmospheric total oxidants is the neutral-buffered KI method. Oxidizing species in the atmosphere react with potassium iodide to release iodine. The iodine may then be measured either spectrophotometrically or coulometrically. Both of these principles have been used in instruments that are suitable for continuous determinations from 0 to 1,000 μg/m^3 (0 to 0.5 ppm) ozone with a sensitivity of about 10 μg/m^3 (0.005 ppm). The spectrophotometric technique also may be used on an intermittent basis provided analysis is completed within 5 minutes after sampling. This limitation is necessary because the iodine color deteriorates with time.

Although currently there is no widely used method for the specific measurement of ozone in the atmosphere, there are several instruments being evaluated for this purpose. These include a device employing the chemiluminescent reaction between ozone and Rhodamine

B and measurement of the emitted light; concentrations less than 2 μg/m^3 (0.001 ppm) can be measured by this technique. Another method uses a gas-phase titration of the air sample with trans-butene-2 to selectively remove the ozone present. This technique is applicable to coulometric or colorimetric methods of iodine measurement, the concentration of ozone in the sample being obtained by difference. Chemical methods for the specific manual determination of ozone incude the 1,2-di-(4 pyridyl) ethylene method, which may be used for 30-minute or 24-hour sampling periods. Though it suffers from several operational drawbacks, this technique is capable of measuring ozone concentrations from 100 to 2,300 μg/m^3 (0.05 to 1.2 ppm) for a 30-minute sample.

The methods generally used for the measurement of PAN include long-path infrared spectroscopy and, more commonly, gas chromatography using an electron-capture detector. Sensitivity of these methods is in the μg/m^3 (ppb) range; the GC electron-capture procedure has been automated by some researchers.

There is a further need for the development of instruments capable of continuously measuring other individual oxidants such as formaldehyde, acrolein, and organic peroxides. Acquisition of data on ambient levels of individual oxidants will facilitate interpretation of the results of studies on the effects of photochemical oxidants.

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CHAPTER 4

EFFECTS ON VEGETATION AND MICROORGANISMS

A. INTRODUCTION

Injury to vegetation was one of the earliest manifestations of photochemical air pollution (1-19). A peculiar type of injury of leafy vegetables, ornamentals, and field crops--characterized by banding, silvering, and stippling of the leaves--was first investigated in the United States by Middleton et al. (12). Plant injury of this kind has since spread to widely separated areas of the world, with increasing severity and with associated economic losses to both farmers and nurserymen (20-29).

Three specific phytotoxic materials have been isolated from the photochemical complex: ozone, nitrogen dioxide, and the peroxyacyl nitrates. The latter homologous series of compounds includes peroxyacetyl nitrate (PAN), peroxypropionyl nitrate (PPN), peroxybutyryl nitrate (PBN), and peroxyisobutyryl nitrate ($P_{iso}BN$). Preliminary studies have shown that PPN is several times as toxic to vegetation as PAN (25,30-32), whereas PBN and $P_{iso}BN$ are more toxic than PPN (30). Since PAN is the only member of the series that has received much study, and since PPN and PBN usually are not measured in the ambient air, discussion will be restricted to the effects of PAN. The presentation of the quantitative effects of ozone and PAN has to be limited to laboratory and controlled field exposures because, under ambient conditions, the effects of these compounds cannot easily be differentiated. The term "oxidant" will be used when discussing the toxic materials to which the plants are exposed under ambient conditions. Research has shown that additional phytotoxicants may be present in the photochemical complex (5,16,33,34). Synergistic effects between the toxicants discussed and other atmospheric contaminants may also produce injury of sensitive plant species (25, 35-39). On the basis of available information, ozone is the most important phytotoxicant of the photochemical complex.

Sensitive plants are useful biological indicators of photochemical air pollution (2,3,9,11,13,14,16,21,33,40-48). Tobacco has been widely used as a monitor for photochemical pollutants (49). Its injury pattern and sensitivity to ozone (50) make it a useful monitor in studying the extent, severity, and frequency of ambient oxidant (41).

Aldehydes and ethylene, which are part of the photochemical complex, also are considered in this chapter. To provide a coherent presentation, it is deemed desirable at this point to consider effects caused by ethylene. This represents a special case, however, because ethylene, though it does enter into the photochemical complex, affects vegetation as a primary pollutant rather than as a result of its photochemical reactions.

Ethylene is a major petrochemical as well as a major product of auto exhaust. Ethylene contributes to the formation of photochemical air pollution and is a significant phytotoxicant (20,51-54). It has been reported to be responsible for considerable losses of both flowers (55) and cotton (56). Ethylene is produced naturally within tissues of plants and serves as a hormone in regulating growth, development, and other processes such as the ripening of fruit (57-59). Thus, ethylene is unique in being both an endogenous plant-growth regulator and a serious phytotoxic air pollutant. While this review was being written, it was learned that several other reviews (60-63) on the same or related topics were also being planned or already under way. These reviews should contain information useful to organizations or individuals interested in air quality criteria for photochemical oxidants or related hydrocarbons in regard to effects on vegetation.

B. CLASSIFICATION OF EFFECTS

The symptoms of air pollution injury of plants attributable to air pollution can be classified into three general categories. *Acute injury* refers to severe visible injury occurring within several hours after exposure; it is identified by cell collapse with subsequent development of identifiable necrotic patterns. *Chronic injury* refers to light to severe injury developing over an extended time period; it is identified by necrotic patterns with or without chlorotic or other pigmented patterns found in sensitive leaf tissues and is not easily attributable to air pollutants other than oxidant. *Physiological effects* include growth alterations, reduced yields, and changes in the quality of plant products. The acute symptoms are generally characteristic of a specific pollutant. Chronic-injury patterns are often highly characteristic but by no means specific for the toxic agents. Disease, insects, nutrition, and other factors can produce leaf patterns similar to those induced by air pollution.

The most easily recognized effect of air pollutants is the necrotic patterns that develop after the occurrence of injury to cells and final collapse of the tissue. The initial plasmolysis is attributable to changes in cell-wall permeability and to changes in structural integrity; a slightly water-soaked or bruised-looking area appears in the intact leaf. These areas usually dry out, leaving necrotic patterns characteristic of the toxicant.

C. SYMPTOMS OF THE EFFECTS OF PHOTOCHEMICAL AIR POLLUTANTS ON VEGETATION

Ozone-type injury to field crops was first observed as a "stippling" on grape leaves in California (64,65). Heggestad and Middleton (66) noted that the tobacco "weather fleck" symptoms found along the eastern seaboard could be reproduced by exposing tobacco to ozone. Daines et al. (67,68) reported ozone-type injury to several crop plants in New Jersey, and indicated that continued production of these crops was seriously threatened. Since these early reports, ozone-type injury to sensitive crop plants has been reported in North America from Florida to southern Ontario (69-77). The conifer "x-disease" (78) and citrus decline in California (26) have also been related to ozone injury.

The initial effect of ozone is in the palisade cell layer and involves scattered groups of cells. In acute injury, the cells plasmolyze and the cell contents become disrupted and disintegrate with or without the production of a dark pigment. These symptoms are called "stipple" in the former instance and "fleck" in the latter. With continuing exposure or with a high-level exposure, these upper-surface necrotic areas will enlarge, coalesce, and eventually spread to the spongy cells and form necrotic lesions through the leaf. Ozone enters the leaf through the stomata, but the gas preferentially attacks the palisade cells. A detailed discussion of ozone symptoms with illustrations of several plants has been published (79).

PAN-type injury, characterized by under-surface glazing or bronzing of the leaves of many plant species, has been observed in California and in the states along the eastern seaboard of the United States (22,25,80). This type of injury has also been reported in other parts of the United States and in several metropolitan areas of other countries (21,28,81-82). Injury occurring in the field has been identified in spinach, beets, celery, tobacco, endive, romaine lettuce, Swiss chard, pepper, alfalfa, petunias, snapdragon, primrose, asters, and other plants (13,14,25,68).

PAN has been accepted as the primary phytotoxicant that causes the oxidant-type injury initially described by Middleton et al. (12), and more completely by Bobrov et al. (3) and Glater et al. (49). The initial collapse is in the spongy cells surrounding the air space into which a stoma opens. The effect in some cases is limited for the most part to cells nearest the lower epidermis. This results in a slight separation of the lower-leaf epidermis, which produces a characteristic under-surface silvering, glazing, or bronzing. More acute injury causes the necrosis to extend through the entire leaf. Injury to the leaves of grasses, petunia, and tobacco causes a cross-leaf banding associated with the sequential maturation of cells from the tip to the base of the leaf. A detailed discussion of PAN symptoms with illustrations has been published (83).

Chronic injury may be characterized by chlorosis or other color changes in addition to several necrotic patterns. Chlorosis, the loss or reduction of chlorophyll, is a very common and nonspecific symptom in plants. Its appearance generally signifies either a deficiency of some nutrient required by the plant or a general metabolic disturbance. The loss of chlorophyll results in leaves with a pale-green or yellow color pattern. There are other color changes in leaves, often with general chlorosis, which are associated with maturity or senescence, e.g., the color change in the autumn. There are instances where in the photochemical complex or its specific components will produce chlorosis in leaves that have been exposed to the pollutant at low levels over a long period of time. The pattern, however, is usually not distinctive, appearing only as an early senescence. The pigmentation of small areas of the palisade cells (stipple) seems to be characteristic of ozone injury in some plants, just as a silvering or bronzing of the undersurface of some leaves is associated with PAN injury.

Physiological effects causing reductions in growth and yield have been experienced in several areas of the United States (25). A serious decline in citrus production, manifested by early loss of leaves, changes in water relationships, smaller fruit, and poor growth (24,26), was experienced in the Los Angeles area. Similar effects were detected in vegetable and other horticultural crops of the area. Photochemical pollutants such as ozone and PAN have been suggested as being responsible (26). Feder and Campbell (84) exposed carnations continuously over a 2-month period to low levels of ozone [100 to

200 $\mu g/m^3$ (0.05 to 0.10 ppm)] and reported a significant reduction in the number of flowers as well as a reduction in height and flower-stock length. The time required to produce flowers was also lengthened. Experimentation has shown that some of these effects, such as early leaf drop, changes in water relationships, increased respiration, subnormal growth of vegetables, and effects on flower development, can be alleviated by exposure to air filtered through activated carbon to remove photochemical oxidants (7,85).

Taylor (86) has reported on observed physiological upsets in trees caused by oxidants. Others have reported on generally poor growth and early senescence due to oxidants (7). Hill and Littlefield (87) have reported a reduction in photosynthesis at ozone concentrations that caused only slight visible injury. A number of articles are available on the subject of metabolic changes in plants exposed to oxidants (88-104). At the present time, there is no sound basis for accurately evaluating the significance of these observations and results.

D. FACTORS AFFECTING RESPONSE OF VEGETATION TO PHOTOCHEMICAL AIR POLLUTANTS (105,106)

The response of a given species or cultivar of plant to a specific air pollutant cannot be predetermined on the basis of the known response of related plants to the same pollutant. Neither can the response be predetermined by the given known response of the plant to similar doses of a different pollutant. Genetic susceptibility and environmental influences must, therefore, be determined for each plant and pollutant.

1. Genetic Factors

Genetic responses account for major variations in the sensitivities of species, as documented in numerous reports. Response variations between varieties, cultivars, or selections within a cultivar have been reported for oat, potato, petunia, spinach, tobacco, white pine, and onion (105). Reports published since 1968 cover the following: horticultural crops (107-113), sweet corn (114,115), petunia (116), forage legumes (117), pine species (118,119), poplars (120), tobacco (121), bluegrass (122), and soybean (123).

Varietal variations have been studied extensively in the species *Nicotiana tabacum*. Heggestad and Menser (50) isolated and developed a sensitive tobacco strain (Bel-W3) and have compared its sensitivity to a number of other varieties. Macdowall et al. (74) tested 32 varieties of tobacco and found the White Gold variety to be most susceptible. Menser (124) and Reinert et al. (110,111) have also studied and reported on varietal response differences in the field and laboratory associated with oxidant or ozone exposures. Injury to susceptible varieties of tobacco has led to the development of more resistant varieties for commercial use. The more sensitive varieties are being used in monitoring programs and as biological material for studying the effects of air pollutants on plants.

Ozone-resistance in onions was found to be related to a dominant gene that controlled the response of the guard cells to ozone (125,126). This single-factor inheritance of sensitivity of onion to ozone is perhaps unique, but it deserves further investigation. Effects on pollen development (127) may be related to reported reductions in fruit yield (114).

2. Environmental Factors

Field and laboratory observations and experiments have shown that the environmental conditions under which plants are grown and/or exposed have a major influence on their sensitivity to phytotoxic air pollutants. Macdowall et al. (73) correlated the sensitivity of tobacco with oxidant levels and found that sensitivity is related to general soil conditions and to the interaction of several meteorological factors such as temperature, wind speed and direction, humidity, and light. By using these factors and an empirical factor (the coefficient of evaporation) and relating them to oxidant dose, Macdowall and his associates had some success in forecasting tobacco-fleck attacks.

During growth, the influence of light intensity on the sensitivity of plants is dependent on the phytotoxicant. Plants have been reported to be more sensitive to PAN when grown under high light intensities (128), but more sensitive to ozone when grown under low light intensities (128-130). Sensitivity of Poa annua during exposure to oxidant shows a positive correlation with increasing light intensity up to at least 32,280 lux (3000 foot-candles) (42). A similar response of plants to ozone has been reported (131).

In a study on the standardization of Poa annua response as an index of smog concentrations, it was found that sensitivity increased as temperatures increased from 4.4° to 29.3°C (40° to 85° F) (42). When plants are exposed under greenhouse conditions, it is almost impossible to separate the effect of temperature at the time of exposure from the effect of light intensity since a positive correlation exists between these two factors. Reported findings are in general agreement that sensitivity of plants to oxidants increases with temperature from 4.4° to 37.7°C (40° to 100°F). However, when plants were exposed to ozone under controlled lighting conditions (131), there was an inverse relationship between temperature and sensitivity as the temperature was raised from 18.2° to 29.3°C (65° to 85°F). This finding suggests that the positive correlation between sensitivity and temperature found under ambient conditions is attributable to the overriding influence of light intensity.

The overall importance of humidity is poorly understood (105). Early work indicated that plants grown and/or exposed under high humidities are more sensitive than those grown under low humidities. Otto and Daines (132) found a marked reduction in sensitivity of pinto bean and tobacco to ozone when the plants were exposed at 26 percent relative humidity as opposed to 51 percent relative humidity. However, the researchers found varied plant responses to higher humidities. These findings agree in general with earlier work. Heggestad et al. (133) noted that tobacco plants grown out-of-doors and in greenhouses in Utah and California were not as sensitive to either ambient oxidant or ozone as similar plants grown in the eastern United States where the relative humidity is higher. Pinto bean and tobacco respond differently to increases in relative humidity from 60 to 80 percent during growth (134). Pinto bean sensitivity to ozone is directly correlated to relative humidity increase, whereas an inverse correlation was found for tobacco.

Even for pinto bean, the humidity response interacted with light intensity in that the relative humidity effect was found only when the growth light was 21,520 lux (2000 foot-candles). No relative humidity effect was found when growth light was 43,040 lux (4000 foot-candles). Relative humidity during exposure is directly correlated with increased sensitivity of plants. This is directly related to water stress. It is possible that a fairly linear response may be found between 10 and 60 percent relative humidity, and maybe higher. Some reports suggest that little effect is found above 70 to 75 percent.

The increased use of carbon dioxide as an additive for increased production in greenhouses may act to reduce the sensitivity to photochemical pollutants of plants grown under these conditions (129).

The relative importance of some meteorological factors, such as wind speed and barometric pressure, has seemed insignificant in relation to the primary factors already discussed. Plants appear to be more sensitive when high pollution levels occur between mid-morning and early afternoon than at other times during the day or night.

Edaphic factors also affect the response of plants to air pollutants (105). Numerous publications relate sensitivity directly to soil moisture stress (135-138). Soil moisture is probably the most significant environmental stress affecting a plant's response to pollutants. It is an important aspect during both growth and exposure to the pollutant. Drought conditions during growth will initiate physiological changes that lead to resistance to environmental stress. Drought conditions during exposure have a direct effect on stomatal opening and thus on pollutant entrance into leaves. Relative humidity and soil moisture together control water stress in plants and are probably inseparable in terms of controlling pollutant effects on sensitive plants. Recent results suggest that O_3 may cause stomatal closure in some plants only if the plants are under water stress (138). Other soil conditions such as nutrition affect plant response to pollutants, but these other factors are of minor importance; the effect of nutrition is significant only under poor cultural conditions or in natural plant communities.

3. Other Factors

The growth stage of a plant is important in determining its sensitivity to air pollutants. Components of the photochemical complex cause different responses in sensitive plants. In annual bluegrass and tobacco, the youngest mature cells are the most sensitive (3,49). Because these cells develop progressively from the tip to the base of the leaf, the injury appears as a band on the sensitive area of the leaf. This pattern is the generally accepted pattern for PAN injury. In other plants, wherein the cells mature rather uniformly within a given leaf, PAN injury affects the young leaves before they are fully expanded. Although the U. S. criteria document indicates that ozone injury generally does not develop until leaves are mature, that document and more recent reports also indicate that ozone effects leaves when they are about 50 to 80 percent expanded--

this being the most sensitive stage (89,130,140,141). In some cases, the stage of leaf maturity is critical with maximum sensitivity occurring over a 2- to-5 day period (130).

There is some evidence that oxidant or ozone injury may be reduced by pretreatment with the toxicant (7,125). In a sensitive onion variety, pretreatment with ozone was linked to stomatal closure that prevented further entrance of ozone. Available information suggests that the stomata of many species are affected by ozone (87).

Little evidence is available on the possible synergistic or antagonistic effects of two or more pollutants in combination. Ethylene, propylene, acetylene, and nitrogen dioxide show no synergistic or antagonistic action; nor do the photochemical phytotoxicants produced by irradiation of nitrogen dioxide-propylene mixtures when ethylene and/or acetylene are mixed with the reaction systems (142).

Interactions between sulfur dioxide and ozone were first studied in the 1950's when sulfur dioxide was injected into an exposure chamber along with the reaction products of ozone-gasoline mixtures (143). Some possible antagonistic responses were suggested in those studies as well as in some more recent ones (144). A synergistic response also was reported by Menser and Heggestad (36); they found that either a 2-hour or 4-hour fumigation with 59 $\mu g/m^3$ (0.03 ppm) ozone or 786 $\mu g/m^3$ (0.3 ppm) sulfur dioxide did not injure tobacco plants, but when plants were exposed to both gases simultaneously at the above concentrations, foliar injury occurred.

Similar results have since been observed in other tobacco cultivars and species (145-147). Macdowall and Cole (148) have also reported that mixtures of ozone and sulfur dioxide enhanced injury in tobacco at concentrations below the threshold for sulfur dioxide, but not below the injury threshold for ozone. An interaction between ozone and sulfur dioxide has been reported to cause injury to white pine (149,150) and has been implicated in the chlorotic dwarf disease of white pine (151) at concentrations of 0.10 ppm or less of each gas. Interactions of ozone and sulfur dioxide also have been reported for as many as 15 to 20 different plant species, including peanut (35), pine (144,152), pinto bean (10), alfalfa, soybean, broccoli, radish, cabbage, and tomato (153).

Only limited studies of the interaction between ozone and sulfur dioxide during continued stress have been reported. Plants studied include radish (38) and tobacco (37). Radish growth was reduced, but the growth reduction was not greater than the additive reductions of the single gases. Tobacco growth was also reduced by the interaction of sulfur dioxide and ozone. In these studies various growth parameters such as fresh and dry weights of top and roots, leaf area, plant height, radish root width and length were measured. The greatest reductions generally were found in the fresh and dry weights of roots.

The interaction of plant-pollutant-pathogen has been studied. Costonis and Sinclair (154) studied the effects of O_3 injury on parasitism of white pine by the fungi, Lophodermium pinastri and Aureobasidium pullulans. With A. pullulans, more invasion occurred when pine needles had been previously injured by ozone. Pine needles injured by ozone before inoculation with L. pinastri had less infection, whereas O_3 injury after infection resulted in more invasion of tissues by the fungus.

Injury to potato from exposure to 294 to 490 $\mu g/m^3$ (0.15 to 0.25 ppm) O_3 increased subsequent infection and invasion by Botrytis cinerea (155,156). Ozone injury to geranium caused 4 to 5 times more infection of flowers by B. cinerea in one variety and 20 to 30 times more infection in another variety.

Several studies show that the effects of plant parasites may suppress injury symptoms from ozone exposures. The mechanism of protection is not unique since three distinctly different disease parasites have produced a similar response. Yarwood and Middleton (19) found that bean and sunflower plants infected with rust fungi were not injured around the rust pustules when exposed to ozone. This effect was not observed until 3 days after infection and was seen beyond the extent of the fungus hyphae. The investigators indicated that a diffusable substance is involved in protecting host tissue. Kerr and Reinert (157) found the same effect in kidney bean leaves showing the halo reaction from the bacterium, Pseudomonas phaseolicola. Brennan and Leone (158) reported that tobacco infected with tobacco mosaic virus was resistant to O_3 injury, whereas non-infected tobacco was severely injured. The physiological basis for these effects is at present unknown.

The mechanism of these effects might provide clues for answering questions concerning the mechanism of O_3 action on higher plant cells.

Relationships between ozone or ambient oxidant and insects have been observed. In California, exposure to oxidant predisposed ponderosa pine to secondary attack by bark beetles (159-162). High insect populations on some pollution-sensitive tree species in the eastern United States may be attributed to the reduced resistance of these species to insect attack because of the effects of pollutants. The combined action of pollutants and insects may produce early senescence and leaf fall as noted in specific species. Observations such as these need to be explored more fully in well-designed experiments. Heagle (163) reviewed this topic in depth and found that some organisms appear to enhance pollutant effects and others seem to prevent or retard pollutant effects.

4. Discussion

The various factors affecting the response of vegetation to air pollution must be mediated through control of the stomata, other internal factors that affect cellular responses, or a combination of these. Although stomatal opening is an accepted prerequisite for the entrance of pollutants, there are certain plant tissues that are sensitive to a specific pollutant and not sensitive to another, even when stomatal function is not a factor. Studies with irradiated auto exhaust best exemplify this fact in that three toxicants were identified from the mixture by the differential responses of tobacco leaf tissue. Biochemical control of sensitivity to ozone and PAN is also suggested, since ozone first affects the mature tobacco leaves and PAN affects the younger leaves, even though stomatal openings are present in leaves of both ages. Thus, although stomatal opening is a prerequisite for pollutant entrance, the injurious reaction of the pollutant within the leaf tissue is a biochemically mediated response, dependent upon unknown factors within the cells.

E. PROBLEMS IN DIAGNOSIS AND ASSESSMENT OF THE ECONOMIC IMPACT OF PHOTOCHEMICAL AIR POLLUTION ON VEGETATION

The injury created by an air pollutant may not only be modified or obscured by environmental factors other than pollution, but the plant may develop injuries from these other factors that are difficult or impossible to distinguish from the injuries caused by air pollution. Ornamental and agronomic crops grown under special management practices must be carefully examined before poor growth is attributed to air pollution. Many bacterial, viral, and fungal diseases, as well as insects, can produce injury symptoms in plants that are quite similar to the symptoms produced by air pollutants. To properly diagnose air pollution effects on vegetation, therefore, the injuries must be observed in the field and supported by both measurements of the pollutant concentration in the ambient air and by laboratory fumigation studies, using different levels of the suspect pollutant. Furthermore, the observer must have a thorough knowledge of local plant-culture conditions.

Although the markings on the leaves of a plant may be associated with an air pollutant, it is often quite difficult to evaluate these markings in relation to their effects on the intact plant. The question that must be answered is whether the plant has been so altered by the air pollutant as to significantly alter its growth, survival, yield, or use. When some of the leaf injuries are evaluated, the problem is relatively straightforward. This is especially true in cases where the appearance of the plant is paramount, or where there is loss of or failure to develop a desired product. Other cases, however, require some assessment of the extent to which the leaf injury has affected the essential processes of the plant. Whether the air pollutants directly or indirectly upset the basic plant processes through injury to the leaf, however, is really immaterial in an evaluation of economic loss.

Guderian et al. (164) expressed a philosophy on the general problem of the effects of air pollutants on vegetation by distinguishing between "injury" and "damage". They defined injury attributable to air pollution as any identifiable and measurable response of a plant to air pollution. They defined damage that results from an air pollution injury as any identifiable and measurable adverse effect upon the desired or intended use or derived product of the plant. Thus, necrotic lesions on the leaves of lettuce caused by an air pollutant are injury. Any assessment of damage requires a judgment that this injury affects the yield or use of the vegetation. The initial identification of injury also requires informed judgment.

Leaf injury to a fruit tree becomes far more difficult to assess as "damage". The effect of a given percentage of leaf destruction on total fruit yield or on the distribution of grades in the crop is not known. It is known that in severe cases the growth of the tree will be affected, yet it is very difficult to evaluate the injury in terms of the value of subsequent crops. To properly evaluate such evidence in relation to a crop that is to be harvested 10 to 20 years later requires more than the knowledge now available. In forest trees, the problem is similar; however, there is evidence of reduced growth-rings that can be attributed to prior leaf injury.

The problem of injury to leaves of ornamentals is equally difficult to assess. If the injury is severe, the stock in a nursery may not be salable. If it is not too severe, pruning may make the stock salable but at a reduced price. The damage in these cases usually can be estimated; however, the problem of injured stock held over for subsequent seasons is more difficult to assess. Often such injured stock may represent a sizable portion of the entire inventory. Then again, individual plants may appear to be fully recovered by the following season. In some cases, if growth of the plant is somewhat stunted, this can be a benefit to the grower.

After the stock has been sold and planted in a yard, park, or cemetery, the concept of damage becomes broader in scope. Damage may not be so much to the leaves of a plant as to the feelings of people concerned.

Though many problems are involved in the evaluation of economic loss, an assessment of economic impact of air pollutants on vegetation in the United States was recently published (165). In summary, the authors estimate that the loss in crops from oxidant air pollution in 1969 was $77 million and that the loss in ornamentals was $42 million. Although these are substantial amounts, they are less than 1 percent of total crop value for the whole United States.

F. DOSE-INJURY RELATIONSHIPS OF PHOTOCHEMICAL OXIDANTS AND VEGETATION

The interrelations of time and concentration (dose) as they affect injury to vegetation (106) are essential elements of air quality criteria. It would be ideal to have a set of standard equations that relate effects to concentrations and duration of exposure with additional terms to account for the influence of the factors that are critical in controlling plant response. Though several equations are discussed later in this section, those equations by no means provide the range of information desired. The open literature contains several papers (130,166-169) in which the relationship between time, concentration, and response is discussed. This includes a publication (29) from Japan (available in English) in which the effects of oxidants on vegetation there are reported and a considerable amount of data is presented.

It is difficult to determine the pollutant dose responsible for affecting plants grown under ambient conditions or to determine the time required to cause such effects. Table 4-1 summarizes some field effects, both chronic and acute, related to average ambient oxidant (ozone) concentrations over varied periods. These data can serve as guidelines for responses that can be expected under variable conditions.

Table 4-2 depicts long-term chronic studies with their respective doses. As the concentration is reduced, a given response not only requires a longer exposure period but a greater dose as well. The results in Table 4-2 are suggestive of what might occur under field conditions. Accumulated days of short-term higher doses may play an important role in producing chronic effects under field conditions.

Table 4-1. EFFECTS OF AMBIENT LEVELS OF OXIDANTS ON VEGETATION (106)

Species	Location	Concentration µg/m^3	Concentration ppm	Time hr	Effects	Reference
Lemon (*Citrus limon*, Burm.)	California	>196	>0.10	Many hours over growing season	32% yield reduction 52% yield reduction	26
Orange (Citrus sinensis, Osbeck)	California	>196	>0.10	148 hrs monthly ave. (March-Oct.); 254 hr monthly avg. (July-Sept.)	54% yield reduction over several years.	
Grape, Zinfandel (*Vitis vinifera*, L.)	California	≥490	≥0.25	Occurred often during 4 month (May-Sept.) growing season	12% reduction in yield the first year and 61% the second year. Decrease in sugar content.	170 171
Corn, sweet (*Zea mays*, L.)	California	392 to 686	0.20 to 0.35	Hourly max. for 3-4 days prior to injury	67% injury in 10 susceptible cultivars (5 cultivars were unmarketable) 18% injury in 13 intermediates 1% injury in 11 resistant cultivars	114
Tobacco (*Nicotiana tabacum*, L.)						
Bel-W3[a]	Maryland	59 to 431	0.03 to 0.22	700 hr	Severe leaf injury	85
Bel-B[a]	Maryland	59 to 431	0.03 to 0.22	700 hr	70% reduction in growth	85
White Gold	Ontario	Dose (conc. x time) of 0.10 to 0.15 ppm-hr			Dose threshold for most sensitive plants (minimal field response)	73
Bel-W3	Ohio	Dose of 0.20 ppm-hr (0.2 ppm for 10 hr)			Gave 50% chance of response	
		39 to 59	0.02 to 0.03	6 to 8 hr	Gave minimal injury response to sensitive monitoring tobacco	129
Pine, Eastern white (*Pinus strobus*, L.)	Ontario	>59	>0.03	10 to 15	Chlorotic fleck of new needles	106

Table 4-1 (continued). EFFECTS OF AMBIENT LEVELS OF OXIDANTS ON VEGETATION (106)

Species	Location	Concentration		Time (hr)	Effects	Reference
		µg/m^3	ppm			
Pine, ponderosa (*Pinus ponderosa*, Laws)	California	>294	>0.15	2 hr/day for 2 months	Chlorotic decline, primarily older needles	172
Bean, white (*Phaseolus coccineus* var. *albus*, Bailey)	Ontario	>157	>0.08	9 hrs	Bronze colored necrotic stipple, premature abscission	77

[a] Not from reference (106).

Table 4-2. EFFECTS OF LONG TERM CONTROLLED OZONE EXPOSURES ON PLANTS(106)

Species	Type of exposure	Concentration		Duration	Effects	Reference
		µg/m^3	ppm			
Duckweed (Lemna minor, L.)	Laboratory	196	0.10	5 hr/day, 14 days	No flowering during exposure; 36% reduction in flowering 1 week after exposure completed Frond doubling rate 7 days in ozone treatment vs. 3 days in control; after exposure recovered to every 4 days Found some injury	173
Carnation (Dianthus caryophyllus, L.)	Laboratory	98 to 176	0.05 to 0.09	24 hr/day, 90 days	50% reduction in flowering, reduced vegetative growth	174
Geranium (Geranium sp.)	Laboratory	372	0.19	9.5 hr/day, 90 days	50% reduction in flowering, flowers have shorter lasting time, reduced vegetative growth	174
Bean, pinto (Phaseolus vulgaris,L.)	Laboratory	255	0.13	8 hr/day, 28 days	79, 73 and 70% reductions in top and root fresh weight and in plant height	175
	Laboratory	98	0.05	24 hrs/day, 3 to 5 days	50% leaf senescence (chlorosis)	176
		98	0.05	24 hrs/day, 5 days	A fivefold increase in lateral bud elongation	
	Laboratory	294	0.15	2 hr/day, 63 days	33% lower plant dry wt., 46% lower pod wt.	177
		490	0.25	2 hr/day, 63 days	95% lower plant dry wt., 99% pod fresh wt.	
		686	0.35	2 hr/day, 63 days	97% lower plant dry wt., 100% pod fresh wt.	

Table 4-2 (continued). EFFECTS OF LONG TERM CONTROLLED OZONE EXPOSURES ON PLANTS (106)

Species	Type of exposure	Concentration µg/m^3	Concentration ppm	Duration	Effects	Reference
Bean, pinto (*Phaseolus vulgaris*, L.) (continued)	Laboratory	294	0.15	2 hr/day, 14 days	8% Results are presented as % reductions of	137
		294	0.15	3 hr/day, 14 days	8% dry wt. of leaves	
		294	0.15	4 hr/day, 14 days	23%	
		294	0.15	6 hr/day, 14 days	49%	
		441	0.23	1 hr/day, 14 days	7%	
		441	0.23	2 hr/day, 14 days	44%	
		441	0.23	3 hr/day, 14 days	61%	
		441	0.23	4 hr/day, 14 days	68%	
		588	0.30	0.5 hr/day, 14 days	11 (9) (% injury on 5 leaves)	
		588	0.30	1 hr/day, 14 days	40 (46)	
		588	0.30	2 hr/day, 14 days	70	
		588	0.30	3 hr/day, 14 days	76 (78)	
Soybean (*Glycine max*, Merr.)	Laboratory	98	0.05	8 hr/day, 5 days/week, 3 weeks	No effect	106
		196	0.10	same time period	24% reduction in root fresh weight; 21% reduction in top fresh weight	
	Field^a	98	0.05	6 hr/day, 133 days	3% reduction in seed yield; 22% reduction in plant fresh wt.; 19% injury	106
		196	0.10	6 hr/day, 133 days	55, 65, 37% values for the above parameters	
Peanut (Arachis *hypogaea*, L.)	Laboratory	39 to 59	0.02 to 0.03	24 to 48 hr or longer	Trace of injury - no yield parameters	35
Corn, sweet (*Zea mays*, L.)	Field^a	98	0.05	6 hr/day, 64 days	9% reduction in kernel dry, wt., 14% injury; 12% reduction in 4 yield parameters	115
		196	0.10	6 hr/day, 64 days	45, 25, 35% values for the above parameters	
	Laboratory	118	0.06	5.5 hr/day, 60 days	Inhibited germination 40%	178
		235	0.12	5.5 hr/day, 60 days	Inhibited germination 90%	

Table 4-2 (continued). EFFECTS OF LONG TERM CONTROLLED OZONE EXPOSURES ON PLANTS (106)

Species	Type of exposure	Concentration		Duration	Effects	Reference
		$\mu g/m^3$	ppm			
Radish (*Raphanus sativus*, L.)	Laboratory	98	0.05	8 hr/day, 5 days/week 5 weeks	54% reduction in root fresh wt; 31% reduction in plant fresh wt; 20% reduction in leaf fresh wt.	38
Pine, white (*Pinus strobus*, L.)	Laboratory	196	0.10	4 hr/day, 5 days/week, 4 weeks	3% needle mottle - evidence that injury occurred over 2-3 days exposure period	179
Pine, ponderosa (*Pinus ponderosa*, Laws)	Laboratory	294	0.15	9 hr/day, 30 days	10% reduction in photosynthesis	172
		588	0.30	9 hr/day, 30 days	70% reduction in photosynthesis	
		882	0.45	9 hr/day, 30 days	85% reduction in photosynthesis	
		294	0.15	9 hr/day, 10 days	4 % reduction in photosynthesis	
		294	0.15	9 hr/day, 20 days	25% reduction in photosynthesis	
		294	0.15	9 hr/day, 30 days	25% reduction in photosynthesis	
		294	0.15	9 hr/day, 60 days	34% reduction in photosynthesis	
		588	0.30	9 hr/day, 10 days	12% reduction in photosynthesis	
		588	0.30	9 hr/day, 20 days	50% reduction in photosynthesis	
		588	0.30	9 hr/day, 30 days	73% reduction in photosynthesis	

[a] Exposed in field chambers.

Several short-term controlled ozone exposures have been followed by injury measurements after a given period of re-growth (Table 4-3). The data from Adedipe et al. (107) reflect pollutant levels that might occur in the ambient air and suggest that injury measurements may give a fair reflection of plant growth reductions resulting from short-term exposures. One must, however, distinguish between growth, which is a rate process (184), and yield. Yield may be reduced when growth is temporarily perturbed by pollutant exposure independently of whether the subsequent growth rate is inhibited (185).

Those interested in ozone effects on deciduous trees may refer to the work of Wood and Coppolino (186) who exposed trees of 21 different species to 196 or 490 $\mu g/m^3$ (0.10 or 0.25 ppm) of ozone for up to 8 hours. Symptoms, relative sensitivity, and age factors are discussed in their report.

Most published data apply to short-term exposures of sensitive plants with resultant acute injury, and even these data are fragmentary. Measurements of response have usually been subjective estimates of visible injury since visible injury is easily recognized and usually fits a subjective scale. An illustrated summary of visible plant injury by pollutants is available (79). The acute effects of the photochemical toxicants have been the most widely studied; the fragmentary reports which are available for PAN are tabulated in Table 4-4, and the relative phytotoxicities of four members of the peroxyacyl nitrates are shown in Table 4-5. Table 4-6 tabulates data on the interaction of sulfur dioxide and ozone.

Table 4-3. EFFECTS OF SHORT TERM CONTROLLED OZONE EXPOSURES ON PLANT GROWTH AND YIELD

Species	Concentration		Time hr	Effects	Reference
	μg/m^3	ppm			
Cucumber (Cucumis sativus, L.)	1960	1.0	1	19% reduction in top dry weight, 4 weeks after exposure at 2 weeks from seed (Ohio Mosaic, cult., 1% injury)	109
	1960	1.0	4	37% reduction in top dry weight (as above, 18% injury)	
Onion (Allium cepa, L.)	392	0.2	24	0% effect	109
	1960	1.0	1	19% reduction in plant dry wt., 4 weeks after exposure at 3 weeks from seed (Spartan era, cult., no injury)	
	1960	1.0	4	49% reduction in plant dry wt. (as above)	
Potato (Solanum tuberosum, L.)	1960	1.0	4 (1 exposure)	0% effect	
	1960	1.0	4 (3 exposures)	30% reduction in tuber dry wt., 3 weeks after final exposure at 4, 5, and 6 weeks after planting (Norland cult., injury not given but was severe)	109
Begonia, white tausendschon (Begonia rex, Putz	196	0.1	2	5% avg. reduction in three growth parameters: 10% shoot weight, 19% flower weight, and flower number	107
	392	0.2	2		
	784	0.4	2	38%	
	1568	0.8	2		
Petunia, Capri (Begonia rex, Putz)	196	0.1	2	9% avg. reduction in three growth parameters (see above)	107
	392	0.2	2	11%	
	784	0.4	2	21%	
	1568	0.8	2	31%	
Coleus, scarlet rainbow (Coleus blumei, Benth)	196	0.1	2	2% avg. reduction in three growth parameters (see above)	107
	392	0.2	2	17%	
	784	0.4	2	24%	
	1568	0.8	2	39%	
Tomato, fireball (Lycopersicon esculentum, Mill)	980	0.5	1	15% reduction in plant wt. (grown under optimum soil	180
	1960	1.0	1	20% reduction in plant wt. moisture conditions)	
	980	0.5	1	15% increase in plant wt. (grown under dry soil	
	1960	1.0	1	25% increase in plant wt. conditions)	
Pine (Pinus strobus, L.)	980	0.5	4	Threshold photosynthesis suppression	181
	1862	0.95	8	Reduced net photosynthesis as much at 80%	182
Soybean	1470	0.75	1	Reduced nodule number and root growth with transitory effect on foliar growth	183

Table 4-4. EFFECTS OF PAN ON VEGETATION

Species	Concentration		Time hr	Effects	Reference
	$\mu g/m^3$	ppm			
Petunia (*Petunia hybrida*, Vilm) sensitive species	495	0.1	5.0	Severe injury to leaves	16
	49	0.01	6.0	Moderate injury	34
Bean (*Phaseolus vulgaris*, L.)	494	0.1	5.0	Moderate injury to 8-day-old leaves	16
	4940	1.0	0.5	100% leaf area injury in plants 5 to 8 days old; no injury in plants 13 to 15 days old	128
	4940	1.0	0.5	Reduced photosynthesis	187
	4940	1.0	0.5	Changes in chloroplasts	188

Table 4-5. RELATIVE PHYTOTOXICITY OF FOUR MEMBERS OF THE PEROXYACYL NITRATES, INDICATED BY PRELIMINARY FUMIGATION ON TWO SPECIES OF PLANTS (30)

		Toxicant							
		PAN[a]		PPN[a]		PBN[a]		$P_{iso}BN$[a]	
Species	Fumigation time. hr	Conc., ppb	Injury, %	Conc., ppb	Injury, %	Conc., ppb	Injury, %	Conc., ppb	Injury, %
Bean, var. Pinto	0.5	--	--	100	90	100	80	100	80
	1.0	140	55	24	7	30	59	--	--
	4.0	40	90	10	86	--	--	--	--
	8.0	20	44	5	100	--	--	--	--
Petunia, var. rosy morn	0.5	--	--	50	7	100	90	25	18
	1.0	140	33	24	35	12	45	--	--

[a] PAN, peroxyacetyl nitrate; PPN, peroxypropionyl nitrate; PBN, peroxybutyryl nitrate; $P_{iso}BN$, peroxyisobutyryl nitrate.

Table 4-6. SUSCEPTIBILITY OF PLANTS TO MIXTURES OF SULFUR DIOXIDE AND OZONE

Species	Concentrations, SO_2/O_3[a]		Time	Effects[b]	Reference
	$\mu g/m^3$	ppm			
Tobacco (*Nicotiana tabacum*, L.)					
Bel-B	2620/196	1.0 /0.1	4 hours	2	153
Bel-W3	655/ 59	0.25/0.03	2 hours	2	36
Bel-W3	262/ 59	0.10/0.03	4 hours	2	25
Bel-W3	655/ 98	0.25/0.05	4 hours	1	153
Bel-W3	2620/196	1.0 /0.1	4 hours	2	153
Bel-W3	1310/ 98	0.5 / 0.05	4 hours	2	153
Bel-W3	655/196	0.25/0.1	4 hours	3	153
Bel-W3	131/ 98	0.05/0.05	8 hours/day - 20 days	1	37
Peanut (*Arachis hypogaea*, L.)	79/ 39	0.03/0.02	6.0 hours	1	35
Tomato (*Lycopersicon esculentum*, Mill)	655/ 98	0.25/0.05	4.0 hours	1	25
Radish (*Raphanus sativus*, L.)	655/ 98	0.25/0.05	4.0 hours	1	25
	262/196	0.1 /0.1	4 hours	2	153
	131/ 98	0.05/0.05	8.0 hours/day - 20 days	1	38
Pine (*Pinus strobus*, L.)	131/ 98	0.05/0.05	8.0 hours/day - 20 days	1 to 2	39
Alfalfa (*Medicago sativa*, L. cv. Vernal)	262/196	0.1 / 0.1	4	1	153
Broccoli (*Brassica oleracea botrytis*, L., cv. Calabrese)	1310/ 98	0.5 /0.05	4 hours	1	153
	262/196	0.1 / 0.1	4 hours	2	153
Cabbage (*Brassica oleracea capitata*, L., cv. All Season)	1310/196	0.5 /0.1	4 hours	2	153
Soybean (Glycine max, Merr.)	131/ 98	0.05/0.05	8 hours/day, 5 days/week, 3 weeks	1 to 2	106
	262/196	0.1 /0.1	6 hours/day, 133 days	2 to 3	106

[a] Concentrations reported for the photochemical complex and ozone have been corrected to neutral KI values. Reported Mast oxidant values were multiplied by a factor of 1.5.

[b] Severity of injury: 1 = slight, 2 = moderate, 3 = severe.

The acute effects of ozone at various concentrations and times has received sufficient study to permit preliminary time-concentration curves to be constructed for several sensitivity groupings. One of the first time-concentration relations was reported by O'Gara (189), who was concerned only with an acute type of injury that develops over a relatively short period of time. He related concentration (c) and time of exposure (T) as:

$$T(c-a) = b$$

The parameters "a" and "b" are dependent upon the species and cultivar of plant and the degree of injury sustained. Concentration is measured in parts per million and time in hours. O'Gara's equation can be rearranged to:

$$c = b/T + a$$

The plot of "c" versus "1/T" is a straight line. The parameter "a" is the intercept for 1/T = 0 or when "T" is infinitely large. This intercept could be considered the threshold concentration for injury. The O'Gara equation is a mathematical form that fits experimental data obtained from exposures limited in time. Guderian et al. (164) do not believe that the O'Gara equation fits their observations and suggested that an exponential relationship best describes their data:

$$T=Ke^{-a(c-r)}$$

where K, a, and r are parameters varying with species and degree of injury. In the middle time range, both forms of equations fit the available data reasonably well. The exponential form probably fits better over a wider range of time.

The acute effects of ozone have received sufficient study to permit the construction of preliminary profiles that relate time, concentration, and response for certain plant species. An adequate expression has been given graphically for pinto bean and Bel W_3 tobacco exposed to ozone (167). Based on this work, a number of plants were exposed to a series to ozone for various times and concentrations and an empirical model was developed to describe the relationship between concentration, time, and response (166). The equation developed was a variation of the early O'Gara equation for sulfur dioxide:

$$C = a + b_1 I + b_2/T$$

where C is the concentration in pphm of ozone, I is the percentage of plant response, T is the time in hours, a is a threshold concept, b_1 is the genetic resistance, and b_2 stands for external resistance factors. This equation permits the development of a true response surface (167) but it forces linearity into the relations between concentration and response, and between time and response, which they do not normally show. The equations also depict a true conceptualization of concentration-time graphs for specific response levels.

In a critical review (106) of the literature for information on dose and response, the authors presented the examples of concentration-response and time-response curves shown in Figure 4-1 and 4-2. The O'Gara equation ($C=a + b/T$) was used in developing the curves. The expected sigmoidal curves were drawn through the actual data points.

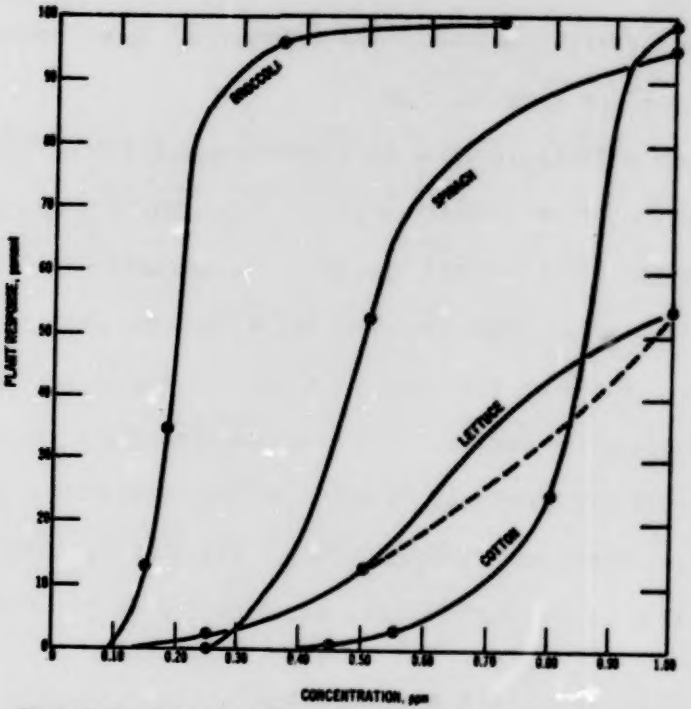

Figure 4-1. Concentration of ozone versus plant response from broccoli, spinach, lettuce, and cotton, for 1-hour ozone exposures. (Results taken from references 130 and 166).

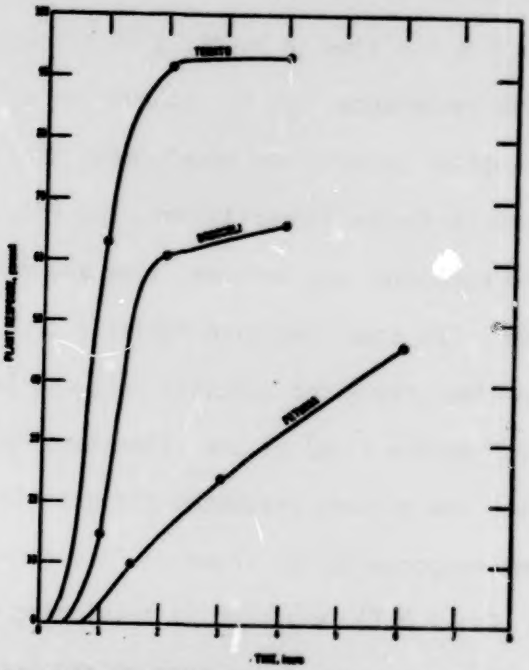

Figure 4-2. Time versus plant response from tomato, broccoli, and petunia to 30 pphm ozone. (Results taken from references 116 and 166).

Over longer times and/or for more resistant species, the curves tend to maximize well below complete injury and may appear more linear than sigmoidal. This should be expected when the whole plant response is determined, but it often happens even when only the more sensitive leaves are considered, as well. Similar curves were plotted, from data obtained from the literature review for over 100 species and varieties. From these latter curves, the scatter diagram for the 5 percent response given in Figure 4-3 was developed. The values from the scatter diagram were used to generate the dose equation shown in the scatter diagrams. The curve shown is an average for many species and varieties of plants. The lower curve (Figure 4-3) is suggested as a threshold curve for responses by sensitive plant species grown

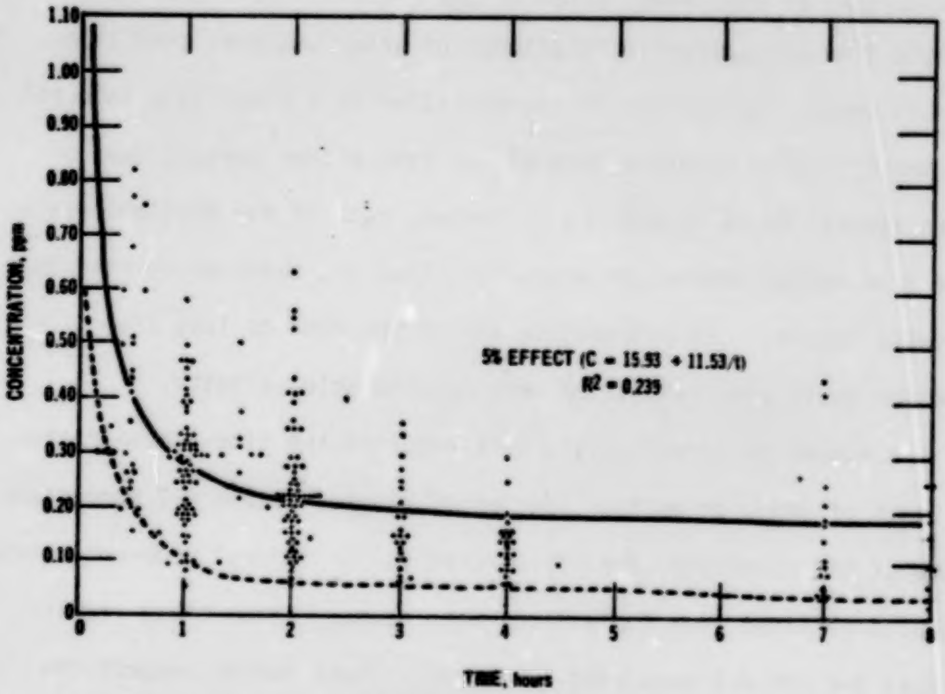

Figure 4-3. Ozone concentration-time scatter diagram of 5% acute plant responses from over 100 species and varieties, calculated from data taken from references, using the equation C = a + b/T and plotting C versus T. (Dotted line represents an approximation of an acute response threshold from data presented, modified by Heck and Brandt (190).

under sensitive conditions.

Such relationships give an insight into what may happen under a given set of circumstances. These relationships are [illegible]ably universal and could be derived for any toxicant producing a defi[illegible]cute-type tissue collapse. Relationships of this type permit the [illegible]tion, with reasonable assurance, that as long as a certain concentration is not exceeded for a given period of time no acute injury will occur. They do not indicate the severity of injury that will occur at higher concentrations for longer time periods. Extrapolation beyond the limits for which experimental data exist is questionable.

None of the experimental data presented provides any more than suggestions of what takes place in the field. These data cover only single time-concentration relationships under standard conditions. They do not cover fluctuations in concentration in a given time interval or the effect of repeated fumigations over either several days or even several hours in one day. Further, much of the available data are from rather short-term exposures, that is, short as related to a growing season. To extrapolate any of the data to long time periods would give results of very questionable validity.

A number of investigators have reported the time-concentration effects of ozone on various species of plants. Table 4-7 summarizes some of the threshold-level (5 percent foliar injury) time-concentration results obtained from fumigations with ozone. From these results, Tables 4-8 and 4-9 have been developed. These tables suggest the durations of exposure and concentrations necessary to produce injury in plants with acute and intermediate sensitivity, and in resistant

Table 4-7. THRESHOLD SUSCEPTIBILITY OF PLANTS TO ACUTE INJURY FROM OZONE

Species	Concentration[a]		Time, hr	Effects[b]	Reference
	µg/m³	ppm			
Tobacco (*Nicotiana tabacum*, L.) Bel-W3	294	0.15	0.5	1	167
	1470	0.75	0.2	1	191
	294	0.15	1.0	1	167
	157	0.08	2.0	2	192
	59	0.03	8.0	1	129
	294	0.15	2.0	2	193
White Gold	96	0.05	3.0	1	73
Bean (*Phaseolus vulgaris*, L.)	294	0.15	0.5	1	167
	784	0.40	0.33	1	43
	157	0.08	4.0	1	25
Oat (*Avena sativa*, L.)	235	0.12	2.0	1	75
White pine (*Pinus strobus*, L.)	196	0.10	4.0	1	69
Alfalfa (*Medicago sativa*, L.)	392	0.20	2.0	2	194
Onion (*Allium cepa*, L.)	784	0.40	2.0	3	125
Peanut (*Arachis hypogaea*, L)	39	0.02	24 to 48	1	35

[a] Concentrations have been corrected to neutral KI values. Reported Mast oxidant values were multiplied by a factor of 1.5.

[b] Severity of injury: 1 = slight, 2 = moderate, 3 = severe

plants at the threshold (5 percent) and 20 percent injury levels, respectively. The lower levels shown in Table 4-8 for the sensitive plants were taken essentially from Table 4-7. The remaining values in Tables 4-8 and 4-9 were developed from the threshold values by extensive study of the effects of ozone on a group of economically important plants.

Table 4-8. PROJECTED OZONE CONCENTRATIONS WHICH WILL PRODUCE, FOR SHORT-TERM EXPOSURES, 5 PERCENT INJURY TO ECONOMICALLY IMPORTANT VEGETATION GROWN UNDER SENSITIVE CONDITIONS

	Concentrations producing injury in three types of plants, ppm[a]		
Time, hr	Acute sensitivity[b]	Intermediate sensitivity	Resistant
0.2	0.35-0.75	0.70-1.00	0.90 and up
0.5	0.15-0.30	0.25-0.60	0.50 and up
1.0	0.10-0.25	0.20-0.40	0.35 and up
2.0	0.07-0.20	0.15-0.30	0.25 and up
4.0	0.05-0.15	0.10-0.25	0.20 and up
8.0	0.03-0.10	0.08-0.20	0.15 and up

[a] Multiply ppm x 1960 to get $\mu g/m^3$ at 25°C and 1 atmosphere.

[b] The lower levels obtained from data presented in Table 4-7.

Table 4-9. PROJECTED OZONE CONCENTRATIONS WHICH WILL PRODUCE, FOR SHORT-TERM EXPOSURES, 20 PERCENT INJURY TO ECONOMICALLY IMPORTANT VEGETATION GROWN UNDER SENSITIVE CONDITIONS

	Concentrations producing injury in three types of plants, ppm[a]		
Time, hr	Acute sensitivity	Intermediate sensitivity	Resistant
0.2	0.40-0.90	0.80-1.10	1.00 and up
0.5	0.20-0.40	0.35-0.70	0.60 and up
1.0	0.15-0.30	0.25-0.55	0.50 and up
2.0	0.10-0.25	0.20-0.45	0.40 and up
4.0	0.07-0.20	0.15-0.40	0.35 and up
8.0	0.05-0.15	0.10-0.35	0.30 and up

[a] Multiply ppm x 1960 to get $\mu g/m^3$ at 25°C and 1 atmosphere

Table 4-10 gives a list of plants which have been studied, places them in three sensitivity classes, and lists them alphabetically by family.

The sensitive range for 0.5, 1, 2, and 4 hours of exposure (Table 4-8) was analyzed using the O'Gara equation. This equation gives a good fit for shorter time periods. These results suggest that the O'Gara equation does not give an exact fit to experimental data with

Table 4-10. SENSITIVITY TO OZONE OF SELECTED PLANT SPECIES[a]

Species	Variety	Reference
Acute Sensitivity		
CHENOPODIACEAE	Early Hybrid	195
Spinach (Spinacia oleracea, L.)		
GRAMINEAE		
Oat (*Avena sativa*, L.), hexaploid		75
Oat (*Avena strigosa*, L.), diploid		75
Rye (*Secale cereale*, L.)		75
LEGUMINOSAE		
Bean (*Phaseolus vulgaris*, L.)	Pinto	
	Sanilac	44,195
		77
Peanut (*Arachis hypogaea*, L.)		35
PINACEAE		
Pine (*Pinus strobus*, L.)		69
POLYGONACEAE		
Smartweed, perennial (*Polygonum* sp.)		195
SOLANACEAE		
Potato (*Solanum tuberosum*, L.)		195
Tobacco (*Nicotiana tabacum*, L.)	Maryland Mammoth	195
	Bel-W3	192
	White Gold	73
Tomato (Lycopersicon esculentum, Mill)	Bonny Best	195
Intermediate sensitivity		
AMARYLLIDACEAE		
Onion (*Allium cepa*, L.)		125

Table 4-10(continued). SENSITIVITY TO OZONE OF SELECTED PLANT SPECIES[a]

Species	Variety	Reference
CHENOPODIACEAE		
Spinach (*Spinacia oleracea*, L.)	Old Dominion	194
	Virginia Savoy	194
	Bloomsdale Long Standing	194
	Hybrid 7	194
Beet (*Beta vulgaris*, L.)		32
COMPOSITAE		
Chrysanthemum (*Chrysanthemum* sp.)	Pippin	195
Endive (*Cichorium endivia*, L.)		32
Stevia (*Piqueria trinervia*, Cav.)		195
CRUCIFERAE		
Broccoli (*Brassica oleracea botrytis*, L.)	Italian Green Sprouting	194
GRAMINEAE		
Annual Bluegrass (*Poa annua*, L.)		32
Barley (*Hordeum vulgare*, L.)	Bonneville	75,194
Sweet Corn (*Zea mays*, L.)	Iochief	194
Field Corn (*Zea mays*, L.)	Portwalco	194
Oat (*Avena sativa*, L.)	Overland	194,195
Rye (*Secale cereale*, L.)		194
Wheat (*Triticum aestivum*, L.)	Lemhi	194
Wheat (*Triticum vulgare*, L.)		75
HYPERICACEAE		
Hypercium (*Hypercium* sp.)		195
LABIATAE		
Coleus (*Coleus blumei*, L.)	Benth	195
LEGUMINOSAE		
Alfalfa (*Medicago sativa*, L.)	Ranger	194
	Unknown	195
Bean (*Phaseolus vulgaris*, L.)	Black Valentine	194
	Mexican Pinto	194
Peanut (*Arachis hypogaea*, L.)	Unknown	195
Pea (*Pisum sativum*, L.)		32

Table 4-10.(continued). SENSITIVITY TO OZONE OF SELECTED PLANT SPECIES[a]

Species	Variety	Reference
PINACEAE		
Pine (*Pinus ponderosa*, Laws.)		196
ROSACEAE		
Peach (*Prunus persica*, L.)	Elberta	194
SOLANACEAE		
Pepper (*Capsicum frutescens*, L.)		32
Tobacco (*Nicotiana tabacum*, L.)	"C"	194
	Turkish	195
	Bel B	169
	Vamorr 48	195
UMBELLIFERAE		
Parsley (*Petroselinum hortense*, Hoffm.)	Moss Curled	194
Resistant		
AMARYLLIDACEAE		
Onion (*Allium cepa*, L.)	Yellow Sweet Spanish	194
	Unknown	125
BALSAMINACEAE		
Impatiens (*Impatiens* sp.)		194
BEGONIACEAE		
Begonia (*Begonia* sp.)		194
CHENOPODIACEAE		
Beet (*Beta vulgaris*, L.)	Detroit Dark Red	194
	Unknown	32,195
COMPOSITAE		
Chrysanthemum (*Chrysanthemum* sp.)		194,195
Endive (*Cichorium endivia*, L.)	Green Curled	194

Table 4-10.(continued). SENSITIVITY TO OZONE OF SELECTED PLANT SPECIES[a]

Species	Variety	Reference
Lettuce (*Lactuca sativa*, L.)	Romaine Unknown	194 32
CONVOLVULACEAE		
Sweet Potato (*Ipomoea batatas*, Lam)	Porto Rico	195
CRASSULACEAE		
Kalanchoe (*Kalanchoe diagremontiana*, Hamet & Pierrier)		195
CRUCIFERAE		
Turnip (*Brassica rapa*, L.)	Purple-Top White Globe	194
Radish (*Raphanus sativus*, L.)	Sparkler, White Tip	194
CURCURBITACEAE		
Cucumber (*Cucumis sativus*, L.)	Marketer	194
GERANIACEAE		
Geranium (*Geranium* sp.)		194,195
GRAMINEAE		
Annual Bluegrass (*Poa annua*, L.)		194
Orchard grass (*Dactylis glomerata*, L)		194
IRIDACEAE		
Gladiolus (*Gladiolus* sp.)	Snow Princess	195
LABIATAE		
Coleus (*Coleus blumei*, L.)		194
Mint (*Mentha piperita*, L.)		195

Table 4-10.(continued). SENSITIVITY TO OZONE OF SELECTED PLANT SPECIES[a]

Species	Variety	Reference
Lettuce (*Lactuca sativa*, L.)	Romaine Unknown	194 32
CONVOLVULACEAE		
Sweet Potato (*Ipomoea batatas*, Lam)	Porto Rico	195
CRASSULACEAE		
Kalanchoe (*Kalanchoe diagremontiana*, Hamet & Pierrier)		195
CRUCIFERAE		
Turnip (*Brassica rapa*, L.)	Purple-Top White Globe	194
Radish (*Raphanus sativus*, L.)	Sparkler, White Tip	194
CURCURBITACEAE		
Cucumber (*Cucumis sativus*, L.)	Marketer	194
GERANIACEAE		
Geranium (*Geranium* sp.)		194,195
GRAMINEAE		
Annual Bluegrass (Poa *annua*, L.)		194
Orchard grass (*Dactylis glomerata*, L)		194
IRIDACEAE		
Gladiolus (*Gladiolus* sp.)	Snow Princess	195
LABIATAE		
Coleus (*Coleus blumei*, L.)		194
Mint (*Mentha piperita*, L.)		195

Table 4-10.(continued). SENSITIVITY TO OZONE OF SELECTED PLANT SPECIES[a]

Species	Variety	Reference
LAURACEAE		
Avacado (*Persea* sp.)		195
LEGUMINOSAE		
Bean (*Phaseolus vulgaris*, L.)	Tendergreen	195
Red clover (*trifolium pratense*, L.)		194
Sensitive plant (*Mimosa pudica*, L.)		195
MALVACEAE		
Cotton (*Gossypium hirsutum*, L.)	Upland 1517	194
	Unknown	197
	Acala SJ-1	130
ONAGRACEAE		
Fuchsia (*Fuchsia* sp.)		194
ROSACEAE		
Strawberry (*Fragaria* sp.)	Empire	195
RUTACEAE		
Lemon (*Citrus limon*, Burm.)		32
SAXIFRAGACEAE		
Piggy-back plant (*Tolmiea menziesii*, Torr & Gray)		195
SOLANACEAE		
Jerusalem cherry (*Solanum pseudocapsicum*, L.)		195
Pepper (*Capsicum frutenscens*, L.)	Grossum Bailey	195
Petunia (*Petunia hybrida*, Vilm.)	Rose Charm	194
UMBELLIFERAE		
Carrot (*Daucus carota*, L.)	Imperator	194
Parsnip (*Pastinaca sativa*, L.)	White Model	194

Table 4-10.(continued). SENSITIVITY TO OZONE OF SELECTED PLANT SPECIES[a]

Species	Variety	Reference
VERBENACEAE		
Verbena (Verbana sp.)	L.A.	195
VITACEAE		
Grape (Vitis vinifera, L.)	Mission	194
	Zing	195
	Tokay	195
	Concord	195
	Unknown	65

[a] Similar and related lists are given in reference 79, 166, and 139.

ozone, but that the approximate fit obtained can be used with a fair degree of confidence with the fragmentary data presently available for analysis.

G. EFFECTS OF PHOTOCHEMICAL OXIDANTS AND OZONE ON MICROORGANISMS

The bactericidal action of ambient photochemical oxidant has been demonstrated by Goetz and Tsuneishi (198). In fact, the Biological Irritation Analogue (BIA) test, in which bacteria of uniform density are exposed to a concentration of oxidant, was developed as a bacteriological analog for the measure of eye irritation from photochemical oxidant aerosols. Photochemical oxidant concentrations from irradiated auto exhaust proved bacteriostatic for common bacteria such as Eschericha coli.

Estes (199) found that exposure of Escherichia coli to an oxidant-type air pollutant mixture inhibited its growth (200). The amount of inhibition was a function of the initial concentration of reactants. The active component in the pollution mixture was isolated and identified as peroxyacetyl nitrate. Glutamic dehydrogenase was isolated from these E. coli cells and exposure of enzyme to the photochemical reaction products resulted in inactivation of the enzyme. Another study (201) suggested that photochemical reactions products of aldehydes would also yield products that inhibited the activity of glutamic dehydrogenase from E. coli. Mudd (202) found that both PAN and ozone oxidized the reduced form of nicotinamide adenine dinucleotide. The reaction product of PAN treatment was biologically active while that from ozone treatment was not. Jacumin et al (203) using Serratia marcescens as test organisms, measured the effect of low concentrations of various pollutants on survival. They found that the photochemical product of 1-hexene and nitrogen dioxide at levels found in polluted air, gave significant killing of bacterial cells. The toxicity of the products of the photochemical reaction in the 1-hexene - NO_2 system was greater than the simple additive effect of the individual components.

Luminescences in bacteria can be adversely affected by exposure to air pollutants. Serat et al (204,205) developed an assay for air pollutants based on the fact that Photobacterium phosphoreum, a luminescent bacterium, was sensitive to photochemical oxidation products. Cells of P. phosphoreum treated with a gas stream containing products formed by the photochemical oxidation of cis-2-butene and nitric oxide lost both luminescence and viability. The rate of luminescence decrease was dependent on the ratio of the initial concentration of reactants. Ozone, nitrogen dioxide, PAN, formaldehyde, and acetaldehyde were also examined in this system. PAN and ozone were effective in decreasing luminescence of *P. phosphoreum*. This organism might be useful in rapidly determining an index of air pollution.

The growth suppression of microorganisms by ozone is a well-known phenomenon (206-214). Ozone at higher concentrations readily oxidizes organic matter and is used in a variety of applications, such as in cold storage plants for the control and suppression of fungi and bacteria associated with food spoilage (215); the purification of drinking water (216-218); and the treatment of sewage (219). The concentrations associated with these uses were 196 μg/m^3 (0.1 ppm) in food protection, 1176 to 3528 μg/m^3 (0.6 to 1.8 ppm) for water purification, and 196,000 to 392,000 μg/m^3 (100 to 200 ppm) for sewage treatment. The value of ozone in preventing the spoilage of fruit and vegetables, however, is in question. The use of ozone with food and drinking water is permitted because safeguards can protect personnel from excessive exposures, but these same safeguards cannot be applied so as to permit the use of ozone for air sterilization in the general ventilation of homes and public buildings.

It is now recognized that ozone is not an effective germicide at concentrations below the level to which humans are sensitive (80 μg/m^3 or 0.04 ppm) (220). The germicidal effectiveness of ozone varies with its concentration, the relative humidity, and the species of bacteria exposed (206,208,221). Jordan and Carlson (221) were unable to obtain any positive germicidal action for ozone against bacteria

(*Staphylococcus pyrogenes*, *Staphylococcus aureus*, and *Bacillus pyocyaneus*) grown on agar plates and exposed to 5880 to 9016 μg/m^3 (3.0 to 4.6 ppm). Concentrations which killed dry typhus bacilli, staphylococci, or streptococci in the course of several hours, were found by Sawyer et al. to kill guinea pigs sooner (207).

Hibben (209) found ozone toxic to the exposed moist fungus spores of some species, even at 200 μg/m^3 (0.1 ppm). Exposure to 980 and 1960 μg/m^3 (0.5 and 1.0 ppm) reduced or prevented germination of spores of all species tested. Ozone at 200 μg/m^3 (0.1 ppm) for 4 hours or at 1960 μg/m^3 (1.0 ppm) for 2 hours stopped apical cell division of conidiophores of *Alternaria solani* and caused collapse of the apical cell wall (210). Studies with *Erisiphe graminis* on barley exposed to 98 to 294 μg/m^3 (0.05 to 0.15 ppm) ozone demonstrated that mycelial growth and sporulation was highly resistant to these ozone levels; however, exposure to ozone at 196, 392, and 588 μg/m^3 (0.10, 0.20 and 0.30 ppm) for 24 hours after inoculation did not affect barley germination, but did reduce infection (222).

Heagle (223) demonstrated reduced uredial growth of *Puccinia coronata* on oats exposed to 196 μg/m^3 (0.1 ppm) ozone continuously 6 hours per day for 10 days; however, urediospores of this parasitic fungus produced on plants exposed to ozone germinated well, produced as many appressoria, and resulted in as much infection as spores produced on unexposed leaves.

Haines (214) found that *E. coli* growth in a culture medium was retarded by 7840 μg/m^3 (4 ppm) ozone in the atmosphere and that 19,600 μg/m^3 (10 ppm) ozone prevented bacterial growth. Elford and van den Ende (224) found that 390 μg/m^3 (0.2 ppm) ozone in a moderately humid atmosphere has a very definite lethal effect on certain bacteria deposited from aerosol mists or suspensoids on various surfaces. The effectiveness depends on 1) the type of surface on which the bacteria are deposited; 2) the medium in which the organisms are contained before being sprayed (a higher protein content favoring protection of the bacterium in the ultimate particle); and 3) the different resistances to ozone of the different types of organism. Relative humidity plays a dominant role in influencing the action of ozone, particularly when the gas is present in low concentrations. In relatively dry air (at relative humidities lower than 45 percent) there was no appreciable killing action on the bacterial aerosols studied, even when the ozone concentration was in excess of 1960 μg/m^3 (1.0 ppm). In contrast, when Streptococcus salivarius, in a fine aerosol-broth suspension, was exposed at a relative humidity of 60 to 80 percent to 50 μg/m^3 (0.025 ppm) ozone for 30 minutes and deposited from the aerosol on glass plate surfaces, a kill of more than 90 percent resulted.

The effects exerted by humidity on fine suspensoid systems of bacteria depend on 1) the purely physical effect of humidity on limiting particle size and settling rate; 2) its effect on normal viability, and 3) the interaction between ozone and the organisms, as reflected in the increased death-rate of the latter in the presence of high humidity. When bacteria are covered with a protective coating of organic matter, as in coarse aerosols from suspensions containing appreciable amounts of serum protein, such as in sneezes and coughs, ozone toxicity rapidly diminishes and much greater concentrations of ozone are necessary to achieve any appreciable killing effect on the organisms. Elford and van den Ende's (224) evidence leads to the conclusion that ozone, at low concentrations which do not otherwise cause irritation of the human respiratory tract, cannot be expected to provide any effective protection against airborne bacterial infection through direct inactivation of the infectious carrier particulates. A summary of the effects of ozone on certain microorganisms is given in Table 4-11.

H. EFFECTS OF HYDROCARBONS ON MICROORGANISMS

The effect of gaseous hydrocarbons on the germination of bacterial spores was studied by Rode and Foster (226). Spore forming bacilli are abundant in all fertile soils. Many of the gaseous hydrocarbons tested were effective inhibitors of spore germination. Among the inhibitors were: propane, *n*-butane, propylene, 1-butene, *trans*-2-butene, 1,3-butene, 3 methyl 1-butene, isobutane, and chloropropanes. Ethylene was slightly effective in inhibiting spore germination but methane and ethane were not. Spore suspensions of *Bacillus megaterium*, *B. subtilis*, *B. cereus* and *B. licheniformis* remained dormant under a partial atmosphere of *n*-butane but germinate when the gas was removed. When pasteurized soil was exposed to gaseous hydrocarbons, the spore formers present in the soil did not germinate until the gas was removed. Studies with *Rhizobium meliloti* (227) demonstrated that the viability of this symbiotic nitrogen fixing organism was affected by exposure to nitrogen dioxide, sulfur dioxide, and aldehydes. The lethal effect of aldehydes was enhanced by high humidity.

Table 4-11. SUMMARY OF EFFECTS OF OZONE ON BACTERIA

	Concentration, ppm	Experimental conditions	Effects	Reference
Bacteria				
Streptococcus salivarius (Organisms sprayed in air in Singleton fine broth suspension and collected on agar plates.)	0.025	30 minutes; relative humidity = 60 to 80 percent; 20°C	90 percent mortality	224
Eschericia (Ozone is admitted when bacteria are inoculated in Nelson's medium.)	4.0		Retarded growth	214
	10.0		Prevented bacterial growth	214
Staphylococcus pyogenes, Bacillus pyocyaneus, Staphylococcus aureus	3.0 to 4.6 (Acid KI Method)	1.5 to 4.5 hours	No positive germicidal action	221
Fungi				
Colletotrichum lindemuthianum	0.1	4 hr/day, 4 days	Spore production; radial growth suppressed	225
Alternaria oleracea	0.0	4 hr/day, 4 days	Spore production increased	225

Several studies have been conducted on the effect of hydrocarbon pollutants on soil fertility. Harper (228) found that natural gas seepage killed the plant cover over the area of leakage. This was shown by Hoeks (229) and Garner (230) to be due to the utilization of all available O_2 from the area during aerobic hydrocarbon degradation resulting in a high degree of anaerobiosis. There was an increase in anaerobic organisms in the soil and in total nitrogen probably due to N fixation by various Clostridia species.

All organic vapor-phase air pollutants eventually are carried onto the surface of the earth. They dissolve in water or attach to plant or soil particles. Abeles et al. (231) studied the fate of air pollutants using ethylene as a model. They estimated that 13.5 x 10^6 MT of ethylene were released as pollutants in 1966 and examined soil for the ability to remove ethylene from the atmosphere. No ethylene uptake occurred in sterilized soil nor in the absence of O_2. There was a 24 hour lag after exposure to ethylene before uptake of the gas by soil occurred. This and the above suggest that microorganisms are responsible for the ethylene assimilation. These workers make a conservative estimate that 6.3 x 10^6 MT of ethylene can be removed from the atmosphere each year by microbial degradation.

I. RELATIVE IMPORTANCE OF HYDROCARBON GASES IN CAUSING INJURY TO VEGETATION

Because ethylene is both a phytotoxic atmospheric pollutant and a growth regulator, the comparative activity of other hydrocarbons as such is of interest. Crocker et al. (232,233) found that exposure to 115 μg/m^3 (0.1 ppm) ethylene for a given time produced epinasty in sensitive plants. The relative concentrations of acetylene, propylene, and 1-butene that produce the same degree of response as ethylene are given in Table 4-12. In this table, the activity of ethylene is defined as 1 with respect to the other hydrocarbons tested. Methane, ethane, propane, butane, 1,3-butadiene, and benzene ring compounds had no effect.

Zimmerman (234) studied growth inhibition of tobacco by ethylene, propylene, acetylene, and 1-butene and found no effects from 1-butene at the highest level used. Burg and Burg (235) found that concentrations of 115 μg/m^3 (0.1 ppm) ethylene inhibited elongation in peas. They

Table 4-12. RELATIVE CONCENTRATIONS OF SEVERAL UNSATURATED HYDROCARBONS THAT PRODUCE BIOLOGICAL RESPONSE SIMILAR TO THAT PRODUCED BY ETHYLENE (234)

		Inhibition of growth		
	Abscission	Pea stem[a]	Tobacco[b]	Epinasty[c]
Ethylene	1	1	1	1
Propylene	60	100	100	500
Acetylene	1,250	2,800	100	500
1-Butene	100,000+	270,000	2,000	500,000
1,3-Butadiene	100,000+	5,000,000	---	--

[a] Reference 235
[b] Reference 236
[c] Reference 232

compared the relative effectiveness of propylene, acetylene, 1-butene, and 1,3-butadiene on the same response. Methane, cis-2-butene, trans-2-butene, and isobutene were inactive.

Abeles and Gahagan (236) found that 115 $\mu g/m^3$ (0.1 ppm) ethylene produced about one-half maximum abscission. This response was then studied using propylene, acetylene, 1-butene, and 1,3-butadiene. These authors tabulated their results along with results of other investigators (232,235,236) to show the relative concentrations of five hydrocarbons that would produce similar activity (Table 4-12).

Heck and Pires (237) fumigated a number of plants with 10, 100, and 1000 ppm of 11 hydrocarbons for periods of 1 to 3 weeks and noted various responses of the test plants. Only ethylene, acetylene, and propylene had adverse effects. The activity of the three gases has been rated (236) in the order noted in Table 4-12. Acetylene and propylene were considerably less effective than ethylene. Methane, ethane, propane, butane, 1-butene, 1,3-butadiene, isobutane, and isobutene were inactive.

From the reports cited, ethylene is the only hydrocarbon that should have adverse effects on vegetation at ambient concentrations of 1150 $\mu g/m^3$ (1 ppm) or less. The hydrocarbon gases most nearly approaching the activity of ethylene are acetylene and propylene, but concentrations of at least 60 to 500 times that of ethylene are required to produce adverse effects. Stephens and Burleson (238) and Stephens et al. (239) reported that ambient concentrations of ethylene in congested urban areas were 25 to 150 $\mu g/m^3$ (0.02 to 0.13 ppm), and propylene concentrations were considerably lower, 5 to 50 $\mu g/m^3$ (0.003 to 0.03 ppm). Ethylene concentrations are, therefore, within the range that will produce harmful effects on vegetation.

J. EFFECTS OF ATMOSPHERIC ALDEHYDES ON VEGETATION

Aldehydes are a major reaction product of the photochemical complex and, as such, have been of some interest to investigators studying the effects of air pollutants on vegetation. Haagen-Smit et al. (240) reported abnormal injury to several sensitive plant species after exposure to fairly high concentrations of several aldehydes, but concluded that injury seen on natural vegetation was not caused by aldehydes. Stephens et al. (16) reported injury to plants from irradiated propionaldehyde but observed no injury where acetaldehyde was a major product from the irradiation of cis-2-butene plus ozone. Hindawi and Altshuller (241) and Altshuller et al. (54) reported injury to sensitive plants after exposure to irradiated propionalaldehyde with and without added nitrogen dioxide, although traces of nitrogen dioxide were present even when not added. They reported no injury after similar exposures to formaldehyde, even though oxidant levels from both aldehydes were sufficiently high to produce plant injury.

Brennan et al. (242) reported injury to petunias that correlated well with total atmospheric aldehyde levels (including ketones), even when oxidant levels were low. This work has been criticized, however, because of the analytical procedure used and laboratory reports on irradiated formaldehyde-nitrogen dioxide mixtures (241).

Results to date suggest that atmospheric aldehydes *per se* are not important phytotoxicants, but that products from the irradiation of propionalaldehyde and higher-molecular-weight saturated aldehydes in the presence of nitrogen oxides may cause injury to plants.

K. SYMPTOMS OF EFFECTS OF ETHYLENE ON VEGETATION

Ethylene enters plant leaves during the course of the normal gas exchange required for growth. For this reason, the site of the primary response is in the leaves, although, in some cases, petals and sepals of flowers, which are actually modified leaves, may be affected. The drying of the sepals, for example is the only acute necrotic change associated with exposure of orchids to ethylene. Responses may range from the death of plant parts to very subtle changes within the leaf cells that can be detected only by biochemical and histological methods.

Chronic injury from ethylene is often nondescript and usually appears as an early senescence of sensitive tissue. It cannot be used to distinguish ethylene from other pollutants or the effects of ethylene from injury caused by other factors such as disease, insects, nutritional disorders, or climatic conditions.

A variety of growth abnormalities, however, have been associated with the exposure of plants to ethylene. In 1901 Neljubow noted that illuminating gas containing about 1145 $\mu g/m^3$ (1 ppm) ethylene caused retarded elongation, an oblique growth toward the horizontal, and a radial swelling or thickening of the stem in seedling plants (51,59,233). These three responses were later termed the "triple response" by Knight and Crocker (243), who suggested that these responses be used to detect the presence of ethylene.

Harvey (53) noted that illuminating gas caused leaves of castor bean to grow in a downward direction and suggested that this epinastic response could also be used to detect ethylene because the plant responded to as little as 115 $\mu g/m^3$ (0.1 ppm). Doubt (52) also reported epinastic growth, as well as leaf abscission, in a variety of plants. Abscission and loss of apical dominance, which results in a spreading vine-like growth instead of an upright plan, have been reported (4,51). Subsequently, the effects of illuminating gas on a variety of plants were studied, and the responses were consistently associated with the ethylene content of the gas (244-247).

Ethylene causes color changes in leaves and flowers and death of flower parts. These responses are in contrast to the effects of other phytotoxic pollutants, which normally cause rather characteristic markings on affected plant leaves, although ammonia can also cause a change in flower color. Most of the reports of adverse effects from ethylene involve flower crops grown in greenhouses (51,248); however extensive injury to a field-grown crop was reported on cotton in 1957 by Hall et al. (56), which was the first such report.

Growth suppression may occur when plants are exposed to dosages of ethylene less than those that cause chronic injury or growth abnormalities. Experiments using carefully controlled air are required to demonstrate this effect, since it is virtually impossible to detect under field conditions. In most cases, growth suppression can be explained on the basis of the effect of the pollutants on biochemical and physiological systems of the plant (249,250).

Characteristic symptoms of ethylene injury include drying of orchid sepals, closing of carnation flowers (sleepiness), and premature dropping (shattering) of snapdragon petals. These symptoms of ethylene pollution cannot be confused with the acute symptoms caused by other pollutants. Chronic symptoms characteristic of natural senescence, however, may be confused with ethylene injury and with certain chronic symptoms associated with sulfur dioxide, ozone, PAN, and several other common pollutants. Accordingly, such effects can not be related to a specific pollutant.

As noted by Brandt and Heck (4), other factors may produce effects that could be confused with ethylene injury. Wilting of leaves caused by water stress, bacterial wilts, or root rots resembles epinasty. These factors, as well as nutritional imbalance and early senescence, cause leaf chlorosis. Furthermore, nutritional imbalance and early senescence could cause premature abscission of leaves and flowers, which is also, as mentioned previously, one of the nonspecific symptoms of ethylene injury.

L. ESTIMATES OF ECONOMIC LOSS (DAMAGE) ASSOCIATED WITH ETHYLENE INJURY TO VEGETATION

The scattered information that exists on economic losses caused by ethylene relates mostly to flower crops. Hall et al. (56) reported that the yield of cotton was meager within 1 mile of an industrial source, but these investigators did not assess the resulting decline in monetary value of the crop. Darley et al. (20) reported that during 1959 the combined damages incurred by three orchid growers in northern California amounted to $70,000.* James (251,252) surveyed losses in the San Francisco Bay area and found that data supplied up to 1964 by several cooperating orchid growers indicated an annual loss ranging from $60,000 to $100,000. Reduction in profit suffered by carnation growers in 1963 was estimated at $700,000. Although information is not available for other parts of the United States, it can be assumed that comparable losses with respect to flower crops could occur in those urban areas where elevated levels of ethylene occur.

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*U.S. Dollars.

For purposes of using plants to assess economic losses resulting from ethylene pollution, the best indicators at present are orchids and possibly carnations and snapdragons. Brandt and Heck (4) have suggested the additional use of cowpea and cotton because of their relatively high sensitivity.

M. DOSE-INJURY RELATIONSHIPS FOR VARIOUS PLANTS EXPOSED TO ETHYLENE

In early investigations, dose-injury relationships for vegetation were studied with illuminating gas and/or ethylene. When comparative tests were made, the concentration of ethylene in the illuminating gas was approximated for parallel experiments with ethylene alone. These experiments were conducted in chambers without air exchange, and concentrations in many experiments ranged from over 1145 mg/m^3 (1000 ppm) to less than 1145 μg/m^3 (1 ppm). Modern analytical techniques were not available, but many early investigators produced valid results and, as noted by Abeles and Gahagan (236), some of these results compare favorably with later work. More recent studies on ethylene have been performed in chambers with dynamic airflow systems and have employed better analytical methods. Concentrations approaching 1145 mg/m^3 (1000 ppm) have been used, but most experiments have utilized lower levels. In general, only the lowest concentrations of the exposure range are considered in the discussion of experimental results in this section.

Because a carnation grower complained that the flowers in his greenhouse did not open normally, or once open would close again, Crocker and Knight (244) investigated this phenomenon and found that ethylene in illuminating gas was responsible. A 3-day exposure to 115 $\mu g/m^3$ (0.1 ppm) ethylene prevented the flowers from opening, and a 12-hour exposure to 575 $\mu g/m^3$ (0.5 ppm) caused the flowers to close, Darley et al. (20) reported that the minimum exposure required to close the flowers was 115 $\mu g/m^3$ (0.1 ppm) for 6 hours.

Zimmerman et al. (246,247) and Crocker et al. (232,233) have exposed a number of plants for various periods of time. Epinasty occurred on the sensitive plants after a 3-hour exposure to 3435 $\mu g/m^3$ (3 ppm) and after a 24-hour exposure to about 345 $\mu g/m^3$ (0.3 ppm). Exposure to as little as 1.15 $\mu g/m^3$ (0.001 ppm) for 20 hours caused epinasty on African marigold, the most sensitive species studied. A 5-day exposure to 1145 $\mu g/m^3$ (1 ppm) produced yellowing along the veins of rose leaves. Of some 202 species tested, 44 percent reacted adversely to ethylene. Long exposure periods were required for abscission of plant parts. Rose leaf abscission began after exposure to 350 $\mu g/m^3$ (0.3 ppm) for 120 hours, while 11,450 $\mu g/m^3$ (10 ppm) induced the same response in 24 hours. Darley et al. (20) found that 115 $\mu g/m^3$ (0.1 ppm) for several hours induced dropping of the flower buds on tomato and pepper plants.

Hitchock et al. (245) reported that all varieties of several species in the lily family were retarded in growth when exposed for 7 days to about 855 $\mu g/m^3$ (0.75 ppm) ethylene contained in illuminating gas. Crocker (51) later reported 25 to 50 percent growth inhibition of four species exposed to 115 $\mu g/m^3$ (0.1 ppm) ethylene for 4 weeks.

Davidson (55) found that when a concentration of 115 μg/m3 (0.1 ppm) ethylene was exceeded for 8 hours, injury (dry sepal) to Cattleya orchids was usually more severe than that normally occurring in greenhouses. At 575 to 1145 $\mu g/m^3$ (0.5 to 1 ppm) for 20 hours, buds remained closed and the tissues collapsed within 2 to 4 days. Dry sepal injury occurred at a concentration of 45 $\mu g/m^3$ (0.04 ppm) for 8 hours and 25 $\mu g/m^3$ (0.02 ppm) for 24 hours. The lowest exposure at which injury occurred was 6 $\mu g/m^3$ (0.005 ppm) for 24 hours.

Darley et al. (20) found the minimum ethylene concentrations for injury to orchids at 1-, 6-, and 24-hour exposures to be 345, 57.5, and 11.5 $\mu g/m^3$ (0.3, 0.05, and 0.01 ppm), respectively. They also fumigated snapdragons and found that exposure to 575 $\mu g/m^3$ (0.5 ppm) for 1 hour caused the petals to fall from the flowers.

Heck and Pires (253) exposed 89 species of horticultural and agronomic crops to the relatively high concentrations of 2290, 5750, and 11,450 $\mu g/m^3$ (2, 5, and 10 ppm) ethylene for 10 days. Based on the severity of a variety of symptoms, they categorized the plants according to six broad groups. Plants typical of the six groups, from the most sensitive to the least sensitive, were cowpea, cotton, squash, soybean, radish, and grasses, respectively. The most marked responses exhibited by cowpea were epinasty, chlorosis, and death of the older leaves. These symptoms later extended to the younger leaves, and death of plants was common. Squash leaves because chlorotic and necrotic, with evidence of growth inhibition, but the plants were not killed. There was no injury to soybean, but growth was retarded. Grasses were not injured and the principal effect observed was the reduction of leaf elongation. Twentytwo of the species flowered during the exposure; floral injury developed on all of them and was quite severe on 18.

The only occurrence of ethylene injury to field-grown crops was reported by Hall et al. (56) on cotton in Texas in 1957. Fields of cotton in the vicinity of a polyethylene manufacturing plant were severely affected, those within 1 mile giving negligible yield because of abscission of the fiber-bearing squares. Lateral buds were stimulated, which resulted in a prostrate, vine-like growth rather than a normal, upright plant. Concentrations of ethylene near the source ranged from 45 to 3435 $\mu g/m^3$ (0.04 to 3 ppm). Injury occurred in the areas having the higher concentrations. In controlled experiments under static conditions, defoliation occurred in 48 to 72 hours with 11,450 to 114,500 $\mu g/m^3$ (10 to 100 ppm).

Heck et al. (254) conducted detailed fumigation experiments on cotton, in which plants were exposed to 690 $\mu g/m^3$ (0.6 ppm) for various periods up to 3 months. Though the plants were seriously affected, the field symptoms noted in the previously reported study were not duplicated, especially the prostrate growth habit. Among the more significant responses after 1 month were an approximately 50 percent reduction in plant height and leaf size, an increase in leaf abscission, an increase in the number of nodes, and an abscission of squares. Leaf chlorosis was not pronounced, but leaves were badly curled and developed a granular texture on their surfaces.

The role of ethylene in fruit dehiscence and young fruit abscission was examined (255). Fumigation of cotton, pecan, and okra fruits with 11,500, 1150, and 115 $\mu g/m^3$ (10.0, 1.0, and 0.1 ppm) ethylene caused marked acceleration of dehiscence. When the role of ethylene in the aging of bean (Phaseolus vulgaris L. cv. red kidney) petiole abscission zone explants was examined, the data indicated that ethylene not only accelerates aging but also induces changes in break strength. It was concluded that ethylene plays a dual role in the abscission of bean petiole explants: a phytogerontological effect and a cellulase-inducing effect (256). A 3-day exposure of bean leaves to 12 to 34 $\mu g/m^3$ of ethylene doubled glucanase activity (257). Ambient levels of ethylene in unpolluted air vary between 1.2 and 6.0 $\mu g/m^3$, whereas in urban areas the levels increase to a maximum of approximately 150 $\mu g/m^3$. Sublethal levels of ethylene can change the biochemical nature of the plant while having no obvious effects on the plant's appearance. Plants grown in the urban environment may be biochemically different from those in rural areas, and the differences may cause problems in comparing research on plants grown in clean air versus those grown in polluted air.

A summarization of the dose-response data is given in Table 4-13. Abeles and Heggestad (258) generalize that threshold effects from ethylene on plants start at 11.5 $\mu g/m^3$ (0.010 ppm), half maximal effects occur at 115 to 575 $\mu g/m^3$ (0.1 to 0.5 ppm), and saturation occurs at 1150 to 11,500 $\mu g/m^3$ (1.0 to 10 ppm). Listings of plants according to sensitivity can be found in several references (4,232,233,246,247, 254).

N. RESEARCH NEEDS

Considerable effort has gone into studying the effects of ozone and the oxidant complex on vegetation, but much more research is

Table 4-13. DOSAGE-RESPONSE RELATIONSHIPS OF VARIOUS PLANTS TO ETHYLENE

Response	Dosage			Reference
	Concentration		Time	
	μg/m^3	ppm		
Abscission				
Cotton leaves, square	46 to 3,435	0.04 to 3.0	Not stated	56
Cotton leaves	685	0.6	1 month	254
Pepper and tomato flower buds	115	0.1	<8 hr	20
Rose leaves	345	0.3	120 hr	247
	11,450	10.0	24 hr	
Snapdragon petals	575	0.5	1 hr	20
Chlorosis on leaves				
Cotton (slight)	685	0.6	1 mo	254
Cowpea	2,290	2.0	1 day	253
Rose	1,145	1.0	5 days	247
Death of plant				
Cowpea	2,290	2.0	10 days	253
Dry sepal injury				
Orchids (severe)	115	0.1	8 hr	55
Orchids (typical)	46	0.04	8 hr	
	23	0.02	24 hr	
Orchids (slight)	5.75	0.005	24 hr	
	345	0.3	1 hr	20
	57.5	0.05	6 hr	
	11.5	0.01	24 hr	
Epinasty				
African marigold	1.15	0.001	20 hr	232
Various plants	345	0.3	24 hr	246
	3,435	3.0	3 hr	
	2,290	2.0	10 days	253
Flowers do not open				
Carnation	115	0.1	3 days	244
Orchid	575 to 1,145	0.5 to 1.0	20 hr	55
Flowers close				
Carnation	115	0.1	6 hr	20
	575	0.5	12 hr	244
Growth inhibition				
Cotton	685	0.6	1 mo	254
	46 to 3,435	0.4 to 3.0	Not stated	56
Lily family	860	0.75	7 days	245
Various plants	2,390	2.0	10 days	253
Loss of apical dominance				
Cotton	46 to 3,435	0.04 to 3.0	Not stated	56

needed.

For example, more research is needed on time-concentration (dose) effects on numerous economically important plants. Information is needed on identification of effects, and the range of effects observed at realistic concentrations and exposure times, including the effects of long-term low concentrations versus short-term peak concentrations.

The effects of exposures to ozone in relation to exposure to other pollutants requires more study. Information is needed on interactions during concurrent exposures and on the effects of exposures in sequence.

Factors influencing quality changes in economic crops need to be better understood. Even though the effects of oxidants on many of these quality changes have been briefly studied, there has been no real effort to correlate the extant studies.

Species competition under oxidant stress needs to be investigated. Although Miller (259) has done such a study in a conifer forest, information on species competition in many communities is needed. In addition, research is needed on oxidant stress in the root rhizosphere resulting from exposure of the aerial vegetative portions of the plant, and on ozone stress on the microflora associated with plants. Further,the effects of oxidants on plant differentiation and growth mechanisms, and on genetic and reproductive structures and fertilization, require study.

There appears to be sufficient evidence available to confirm the belief that ethylene is the only hydrocarbon that affects vegetation at concentrations known to be present in ambient air. To produce comparable effects, the other active gases, acetylene and propylene, must be present in concentrations that are at least 100 times that of ethylene. The range of plant responses induced by ethylene is known and has been confirmed by many investigators. Since the principal effect at reported ambient concentrations is on plant growth, more information is needed concerning the long-term economic effects of concentrations lower than 345 $\mu g/m^3$ (0.3 ppm) on a wide variety of greenhouse and field crops. Heck and Pires (253) examined a large number of crops exposed to ethylene, but the concentrations were relatively high and the exposure to ethylene was for only 10 days. Heck et al. (254) observed a significant reduction in the growth of cotton at 690 $\mu g/m^3$ (0.6 ppm) over a period of 1 to 3 months, but were able to examine only the one species. These results indicate the direction that continued research should take.

The possible synergistic action of ethylene in combination with other pollutants, particularly those occurring in the photochemical complex, should be investigated. Heck (142) observed no synergistic effect on several species; however, Menser and Heggestad (36) reported that a mixture of ozone and sulfur dioxide caused injury to tobacco when the same concentrations acting alone had no effect. Consequently, research in this area is also needed.

O. SUMMARY

Sensitive plants have been useful biological indicators of photochemical oxidant pollution. The acute symptoms of plant injury are generally characteristic of a specific pollutant while, though highly characteristic, chronic injury patterns are not. Injury to leaves by ozone appears as a stippling or flecking of the leaves. Such injury has occurred experimentally in the most sensitive species after exposure to 60 $\mu g/m^3$ (0.03 ppm) ozone for 8 hours. Injury will occur in shorter time periods when low levels of sulfur dioxide are present. PAN-produced injury is characterized by an under-surface glazing or bronzing of the leaf. Such injury has occurred experimentally in the most sensitive species after exposure to 50 $\mu g/m^3$ (0.01 ppm) PAN for 5 hours. Leaf injury has occurred in certain sensitive species after a 6- to 8-hour exposure to 39 to 59 $\mu g/m^3$ (0.02 to 0.03 ppm) of total oxidant, which includes ozone, PAN, and other pollutants. Ozone, however, appears to be the most important phytotoxicant in the photochemical complex.

There are a number of factors affecting the response of vegetation to photochemical air pollutants, such as light intensity, relative humidity, and the age of the plants or its parts. Variability in response is known to exist between species of a given genus and between varieties within a given species. Varietal variations in sensitivity have been most extensively studied with tobacco and several other plants. In fact, a sensitive tobacco strain, Bel-W3, has been isolated and developed for use as a biological indicator of ozone injury.

The influence of light intensity on the sensitivity of plants during growth appears to depend on the phytotoxicant. Plants have been reported to be more sensitive to PAN when grown under high light intensities, but more sensitive to ozone when grown under low light intensities. Reported findings are in general agreement that the sensitivity of greenhouse-grown plants to oxidants increases with temperature from 4.4 to 32.7°C (40° to 100°F). However, there is some indication that this positive correlation may result from the overriding influence of light intensity on sensitivity.

The effects of humidity on the sensitivity of plants has not been well documented. General trends indicate that plants grown and/or exposed under high humidities are more sensitive than those grown at low humidities. Though there has been little research in this direction, there are indications that soil factors influence the sensitivity of plants to phytotoxic air pollutants. For example, plants grown under drought conditions are less susceptible than those grown under moist conditions.

The age of the leaf being exposed is important in determining the sensitivity of the leaf to air pollutants. There is some evidence that oxidant or ozone injury may be reduced by pretreatment with the toxicant.

Identification of an injury to a plant as being caused by air pollution is an arduous undertaking. Even when the markings on the leaves of a plant may be identified as injury from an air pollutant, it is often quite difficult to evaluate these markings in terms of their effect on the intact plant. Further difficulty arises in trying to relate such markings to the economic losses caused by air pollution damage to the plant.

The interrelations of time and concentration (dose) as they affect injury to plants are essential considerations in the establishment of to air quality criteria. There are, however, only scant data relating concentrations and duration of photochemical oxidant exposure to chronic injury and effects on reductions in plant growth, yield, or quality. There is also a dearth of information relating acute injury to concentrations and duration of exposure to PAN or mixtures of photochemical oxidants. A larger body of information exists on the acute effects of ozone, but, even in this instance, the information is far from complete.

Bacteriostatic and bactericidal properties of photochemical oxidants in general have been demonstrated. The growth suppression of microorganisms by ozone is a well-known phenomenon, although the ozone concentrations at which suppression occurs are detrimental to humans. The bactericidal activity of ozone varies with its concentration, the relative humidity, and the species of bacteria exposed.

Hydrocarbons were first recognized as phytotoxic air pollutants, about the turn of the century, as a result of complaints of injury to greenhouse plants from illuminating gas. Ethylene was shown to be the injurious component. Renewed interest in hydrocarbons, and ethylene in particular, occurred in the mid-1950's when ethylene was found to be one of the primary pollutants in the photochemical reaction complex. Research on several unsaturated and saturated hydrocarbons proved that only ethylene had adverse effects at known ambient concentrations. It is noteworthy that the phytotoxicity of acetylene and propylene resemble more closely that of ethylene than do other similar gases, but 60 to 500 times the ethylene concentration is needed to produce comparable effects with these gases.

In the absence of any other symptom, the principal effect of ethylene is to inhibit the growth of plants. Unfortunately, this effect can not be characterized as ethylene injury only because other pollutants at sublethal dosages, as well as some diseases and environmental factors, may also inhibit growth.

Epinasty of leaves and abscission of leaves, flower buds, and flowers are somewhat more typical of the effects of ethylene, but the same effects may be associated with nutritional imbalance, disease, or early senescence. Perhaps the most characteristic effects of ethylene are the dry sepal wilt of orchids and the closing of carnation flowers. Injury to sensitive plants has been reported after exposure to ethylene concentrations of 1.15 to 575 $\mu g/m^3$ (0.001 to 0.5 ppm) for 8 to 24 hours.

Economic loss from ethylene damage to plants has not been widely documented except among flower growers in California, where damage to orchids and carnations has been assessed at about $[illegible]00,000 annually. More research needs to be done to permit evaluation of economic losses sustained in field and greenhouse crops from long exposures to very low concentrations of ethylene.

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CHAPTER 5

EFFECTS OF OZONE ON MATERIALS

A. INTRODUCTION

Ozone is a major factor in the overall deterioration of a number of different types of organic materials. In fact, certain specific organic compounds are more sensitive to ozone attack than are humans or animals. The magnitude of damage is difficult to assess in some cases because naturally occurring ozone is a component of weathering and weathering itself is a major cause of materials deterioration. Nevertheless, researchers have shown that atmospheric ozone resulting from the activities of man has been responsible for accelerating the deterioration of several classes of materials.

B. MECHANISMS OF OZONE ATTACK

Ozone is so active that, when concentrated, handling it becomes a problem because of its effect on ordinary materials. In general, any organic material is affected deleteriously by concentrated ozone (1). Bailey (2) has reviewed the literature from 1939 to 1957 thoroughly for reactions of ozone with organic compounds, and he describes the reaction mechanisms in detail. Although it is incorrect to assume that all of these reactions will occur at atmospheric concentrations of ozone, it is possible that some of these mechanisms would be operant.

Many polymers are sensitive to atmospheric concentrations of ozone (3). Both chain-scissioning and cross-linking take place in polymers exposed to ozone at atmospheric levels. Chain-scissioning results in molecular weight changes and losses in tensile strength.

The sensitivity of polymers to chain-scissioning, therefore, is related to the prevalence of double bonds in their structure. Cross-linking increases the rigidity of the polymer, reducing elasticity and increasing brittleness.

C. ELASTOMERS

The chemical structures of many elastomers contain double bonds and these elastomers are therefore, prone to oxidative reactions and especially to ozone attack (4,5). The most vulnerable generic elastomeric materials are natural rubber and synthetic polymers of styrene-butadiene, polybutadiene, and polyisoprene (6). These elastomers account for 87 percent of 1972 United States production (7). Except for natural rubber, tires are the major use for these elastomers (8). Butyl, halogenated butyl, polychloroprene, vinyl-modified nitrile-butadiene, and carboxylated nitrile elastomers have some ozone resistance but require special formulation for optimum performance. Synthetic elastomers with saturated chemical structures, such as silicones, ethylene-propylenes, chlorosulfated polethylenes, polyacrylates, and fluorocarbons, have inherent ozone resistance (4, 9-15). These latter special-application materials, however, are relatively expensive and account for only a small fraction of the market on a weight basis.

Tensile stress is necessary to produce ozone-cracking in sensitive elastomers. If strained as little as 2 or 3 percent and exposed to an atmosphere containing 20 to 40 $\mu g/m^3$ (0.01 to 0.02 ppm) ozone, cracks will develop perpendicular to the stress axis (10,11). Elastomers in a relaxed state can be exposed for long periods of time to relatively high concentrations of ozone without developing visible cracking (16). The elastomer is apparently protected by the formation of an ozonide film on the surface (4). The embrittled film cracks when strained and exposes fresh surface to the atmosphere. The strong dependence of crack-growth on nominal tensile stress has been explained in terms of the Griffith theory of fracture mechanics (17). The Griffith theory, however, only partially explains observed relationships between number and size of cracks as a function of strain (5,17). Crack-growth rate is also ozone-concentration-dependent (17), which suggests that the rate of diffusion of ozone to the root of a crack is the rate-controlling factor.

Andrews (18) has taken advantage of the fracture mechanism behavior of various elastomer to develop formulations that possess ozone resistance and other desired properties. Blends of natural rubber with an ethylene-propylene copolymer are more resistant to ozone-cracking than is natural rubber alone. Specimens of blends from 0 to 50 percent ethylene-propylene, strained from 5 to 100 percent, were exposed to 980 mg/m^3 (500 ppm) ozone in air. Microcracks were observed in all of the blends, but the crack density increased with a decrease in ethylene-propylene copolymer content. The addition of the ethylene-propylene copolymer raises the critical stored energy necessary to propagate the observed microcracks into the macrocracks characteristic of ozone damage.

Antiozonant additives, such as p-phenylenediamine derivatives, have been developed and protect elastomers from ozone degradation. These chemicals are expensive, however, and add to the cost of elastomeric products (19). The addition of 1.5 percent antiozonants to automobile tires costs up to $0.50 per tire in the United States. Antiozonants are also used in conveyor belts, automotive parts, wire and cable insultation, and other products.

Antiozonants, however, have limitations. The amount necessary for effective control increases with the anticipated concentration of atmospheric ozone (20). Furthermore, antiozonants tend to migrate to the surface of products, where subsequent usage may remove them. Oils, gasoline, and other chemicals may also extract them (4). In these cases, antiozonants provide only temporary protection against ozone damage. Thus, in spite of preventive measures, ozone cracking of elastomeric products remains a major problem.

A number of factors affect the rate of attack by ozone on elastomers (4): the amount of tensile stress, type of elastomeric compound, concentration of ozone, period of exposure, rate of diffusion of ozone to the elastomer surface, and temperature. Dose-response data, therefore, are necessarily dependent on these factors. Methods for reporting damage vary from one researcher to another, further complicating comparison. For example, damage may be expressed as time to initate either microcracks or visible cracks, or it may be expressed in crack-depth or crack-growth rate. Dose-response data should be considered with these factors in mind.

Much of the documented research on ozone-elastomer effects has been conducted with ozone concentrations above those normally existing in polluted atmospheres. This has been intentional in order to accelerate damage reaction rates. Nevertheless, investigators have carried out some research at lower ozone concentrations.

Bradley and Haagen-Smit (12) evaluated a natural rubber formulation for susceptibility to ozone-cracking (Table 5-1). This particular formulation was selected for its sensitivity to ozone. Strips were strained approximately 100 percent by bending. These specimens, when exposed to 39,000 mg/m^3 (2 percent or 20,000 ppm) ozone in air, cracked instantaneously and broke completely within 1 second; however, when they were exposed to various low concentrations of ozone, different time periods were required for cracks to develop (Table 5-2). A log-log plot of ozone concentration versus cracking time yields a straight-line correlation, thus indicating exponential behavior. Such behavior is predictable since it is often observed in various elastomer aging processes.

Edwards and Storey (20) have determined the effects of ozone on "hot" (Polysar-S) and "cold" (Polysar-Krylene) stryene-butadiene (SBR) elastomers containing various amounts of antiozonants. These basic elastomeric formulations were used in tire sidewalls (Table 5-3). The investigators exposed test specimens under stress (100 percent) to an ozone concentration of 490 ± 100 μg/m^3 (0.25 ± 0.05 ppm) at 49°C (120°F). Table 5-4 presents test results that show significant reductions in rate of cracking produced by increased amounts of antiozonants.

Meyer and Sommer (21) exposed thin polybutadiene specimens, under constant load, to room air for which the average concentrations of ozone had been determined. Specimens exposed in the summer months to average ozone concentrations of about 94 μg/m^3 (0.048 ppm) failed by

Table 5-1. FORMULATION OF HIGHLY OZONE-SENSITIVE RUBBER (12)[a]

Ingredient	Parts by weight	Percent by weight
Rubber	100	35.91
Tire reclaim	125	44.88
SRF Black	33	11.85
Stearic acid	1.5	0.54
Pine tar	8.4	3.02
Zinc oxide	4.7	1.69
Mercaptobenzothiazole	0.8	0.29
Diphenylguanidine	0.1	0.03
Sulfur	5	1.79

[a] Cured 40 minutes at 310,050 N/m^2 (45 psi) steam.

Table 5-2. EFFECT OF OZONE ON RUBBER (12)[a]

Ozone concentration[b]		Time to first sign of crack at 4X magnification, minutes
$\mu g/m^3$	ppm	
40	0.02	65
510	0.26	5
880	0.45	3

[a] Special formulation of Bradley and Haagen-Smit, strained 100 percent.

[b] Determined by neutral KI.

Table 5-3. TIRE SIDEWALL FORMULATION (20)

Ingredients	Parts by weight
Polymer (hot or cold SBR)	100
Circosol 2 XH	10
FEF Black	30
SRF Black	10
Zinc oxide	3
Stearic acid	2
Antiozonant (Santoflex AW)	Variable
Crystex	2

Table 5-4. EFFECTS OF OZONE ON SIDEWALL FORMULATIONS CONTAINING VARIOUS ANTIOZONANT CONCENTRATIONS (20)[a]

Polymer	Antiozonant concentration, (Santaflex AW), percent	Rate of cracking		Time to first sign of crack, minutes[b]
		10^{-4} cm/hr	10^{-4} in /hr	
"Hot" SBR (Polysar-S)	0	2.34	0.92	65
	0.32	1.75	0.69	87
	0.63	0.89	0.35	170
	1.25	0.33	0.13	460
"Cold" SBR (Polysar-Krylene)	0	4.01	1.58	38
	0.32	2.16	0.85	71
	0.63	1.45	0.57	105
	1.25	0.61	0.24	250

[a] Specimens were strained initially at 100 percent and exposed at 49°C (120°F) to an ozone concentration of 490 ± 100 $\mu g/m^3$ (0.25 ± 0.05 ppm).

[b] Added to enable comparison with data in Table 5-2. First sign of crack was assumed to be 2.54 μm (10^{-4} inch) crack depth, visible at 4X magnification. Data are not found in reference 20.

breaking into two separate parts after 150 to 250 hours. In the fall, at average ozone concentrations of 82 μg/m^3 (0.042 ppm), specimens failed between 400 and 500 hours. In the winter, at average ozone concentrations of 47 μg/m^3 (0.024 ppm) failures occurred between 500 and 700 hours. These data show the strong dependence of cracking rate on average concentrations of ozone.

The behavior of rubber exposed to ozone under laboratory conditions correlates well with the service behavior of tires in localities where atmospheric ozone concentrations are high (22). The relative susceptibility of white sidewalls made from different formulations remains the same, whether in a laboratory test, in which they are exposed to as much as 980 μg/m^3 (0.5 ppm) ozone, or in the ambient air of the Los Angeles area. The rate of cracking is thus a function of ozone concentrations.

As the tread wear on passenger car tires improves, more or better antiozonants will have to be added to sidewall formulations to prevent sidewall cracking from becoming the limiting factor in tire life. Thus, part of the cost of premium tires will be the result of increased protection against atmospheric ozone.

Mueller et al. (23,24), on the basis of a study completed in 1970 to assess the economic impact of air pollution on elastomeric products in the United States, estimated loss at the consumer level of $500 million annually. About $170 million of this total was associated with various means of protection, including antiozonants, special polymers, and waxes.

D. TEXTILES

Cellulose fibers are vulnerable to oxidation; therefore, Bogaty et al. (25), therefore, carried out experiments to study the potential role of ozone in the deterioration of cotton textiles. They exposed samples of duck and print cloth to air containing between 40 and 120 $\mu g/m^3$ (0.02 and 0.06 ppm) ozone at room temperature and in the absence of light. Samples were exposed both dry and wet; the moisture content of the wet samples was never less than 50 percent. The researchers used changes in fluidity values to assess degradation. Fluidity (reciprocal of viscosity) is an indirect measure of a polymer's average molecular weight; an increase in fluidity indicates a reduction in molecular weight. Such reductions reflect changes in tensile strength and other physical properties.

After the cloth samples were exposed for 50 days, fluidity values increased for the cotton samples that were moist during exposure, but did not appreciably change for the samples that remained dry during exposure. Fluidity values for control samples that were kept moist but not exposed to ozone showed little change. These results indicate that ozone caused virtually all the degradation. In addition to increased fluidity, the moist samples had a 20 percent loss in tensile strength after exposure to ozone. Similar fabrics were also exposed to higher levels of ozone, resulting in a greater increase in degradation (fluidity) for the moist samples. Based on these results, low levels of ozone will degrade cotton fabrics if they are sufficiently moist. The mechanism of ozone attack appears to be primarily through solution in water, since at room temperature ozone, compared to air, is considerably more soluble in water.

The investigators estimated that the laboratory exposure conditions used (50 days, high moisture and ozone) were equivalent to 500 days exposure under field conditions. They concluded that while ambient levels of ozone are capable of damaging moist cotton fabrics, the deterioration is slight in comparison with that from other elements of weathering such as light, heat, alternate wetting and drying, and microorganisms.

Morris (26) also studied the effects of ozone on cotton. Special print cloth fabric samples were exposed in the absence of light to 995 $\mu g/m^3$ (0.5 ppm) ozone for 50 days in a chamber maintained at 21°C (70°F) and 72 percent relative humidity. No appreciable effect upon breaking strength or fluidity measurements was found. Apparently the moisture content of the cotton (9 percent) was not high enough to produce the degradation that Bogaty measured in wet cotton samples even though the concentration of ozone was considerably higher (about 10 times).

The laboratory study of Kerr et al. (27) examined the effect of periodic washing on the tendering of cotton fabrics exposed to ozone. They exposed samples of print cloth, dyed with C. I. Vat Blue 29, in a chamber operating at room temperature, in the absence of light, and continuously supplied with clean air contaminated with ozone having an average concentration of 1470 $\mu g/m^3$ (0.75 ppm). The investigators did not measure relative humidity although they attempted to increase the humidity by placing a pan of water on the chamber floor. The cotton samples were removed from the chamber at 3-day intervals. Half of them were machine-washed and the other half were soaked in water for 1 minute. All samples were passed through a hand wringer to remove excess water before they were returned to the chamber for further exposure. Control samples were kept in a light-tight box maintained at 21°C (70°F) and 65 percent relative humidity and were given the same washing and soaking treatment.

After an exposure of 60 days, which included 20 washing or soaking treatments, the change in strength of the control fabrics was not significant. By comparison, the fabrics exposed to ozone changed significantly; the loss in strength for the washed fabrics was 18 percent and for the soaked fabrics, 9 percent. The investigators attributed these losses to ozone.

Since Morris (26) found no degradation under exposure conditions similar to those used by Kerr et al., it appears that the washing and soaking treatment in some way affects the sensitivity of the fabrics to ozone. Nevertheless, when one attempts to equate these findings with "real-world" conditions, the degradation seems minimal in view of the fact that average levels of ozone under field conditions are less than 10 percent of the levels used in the laboratory exposure.

In recent laboratory studies, Zeronian et al. (28) simultaneously exposed modacrylic (Dynel), acrylic (Orlon), nylon 66, and polyester (Dacron) fabrics to artificial sunlight (xenon arc) and charcoal-filtered clean air contaminated with 365 $\mu g/m^3$ (0.2 ppm) ozone at 48°C (118°F) and 39 percent relative humidity. During exposure, the fabric samples were sprayed with water for 18 minutes every 2 hours. Ozone damage was measured by comparison with fabrics exposed to the same environmental conditions but without ozone. After exposure for 7 days, Zeronian et al. found that ozone did not affect the modacrylic and polyester fibers, but appeared to affect slightly the acrylic and nylon fibers, although the evidence was not clear.

E. TEXTILE DYES

Although atmospheric nitrogen oxides had been identified during the early 1900's as an important cause of color fading in certain textile dyes, mainly disperse dyes on acetate fabrics, it was not until the mid-1950's that the research of Salvin and Walker (29) showed that atmospheric ozone is also an important cause of dye fading. The discovery came about as a result of field testing acetate fabrics containing newly synthesized blue disperse dyes. Previous laboratory exposures of these dyes had shown them to be highly resistant to fading by nitrogen oxides. Field testing was carried out to determine if the performance of these new dyes was as good in actual use as in laboratory tests.

The researchers exposed fabrics dyed with one of the new blue dyes (as a color component) in homes located in areas known to have either low or relatively high levels of nitrogen oxides. At the low nitrogen oxides sites, they found that fading for many of the fabrics was equal to, and in some cases exceeded, the fading observed at the higher nitrogen oxides sites. Much of the observed fading was characterized by a bleached, washed-out appearance rather than the familiar reddening that nitrogen oxides produce in most sensitive dyes. This anomalous behavior suggested that some other oxidizing agent was responsible and was present at all exposure locations.

To describe this fading phenomenon, Salvin and Walker coined a new term, "O-fading." Follow-up laboratory investigations and general observations showed atmospheric ozone to be responsible. Ozone concentrations of 200 $\mu g/m^3$ (0.10 ppm) produced marked fading in most of the blue disperse dyes and some of the reds and yellows.

The discovery of O-fading was useful in helping explain much of the anomalous fading of certain dyed fabrics observed during subsequent light-fastness testing and service trials (30-33). Other types of dyes besides the disperse dyes showed abnormal fading. Furthermore, the service trials pointed out the importance of relative humidity, suggesting that the resulting increased moisture content of fibers such as cotton would promote and accelerate the absorption and reaction of pollutants with vulnerable dyes. The service trials also alerted the textile industry to the possibility of potential consumer complaints. Such complaints began to appear in the early 1960's and were mainly concerned with two distinctly different textile materials: polyester/cotton permanent press fabrics and nylon carpets.

Fading of polyester/cotton permanent press fabrics was noted first on the folds and edges of slacks stored in warehouses or on the stock shelves of retail outlets (34,35). Some garments faded after storage in warehouses for only 10 days. Incidents occurred in various locations including California, Texas, and Tennessee. Because of the volume of garments involved, some producers suffered heavy economic losses. Fading was marked by a loss in blue along with a slight increase in red. This suggested that ozone and, to a lesser degree, nitrogen oxides may have been the active fading agents since sunlight was obviously not a factor. However, this did not seem reasonable because the cotton was dyed with vat dyes and the polyester with disperse dyes; both dye-fiber systems are generally resistant to fading by common air pollutants. Furthermore, fading occurred only on fabrics that had been made into finished products and cured to set the permanent press resin; fading had not been observed during the temporary storage of dyed fabrics either before or after treatment with permanent press resins, prior to curing.

After a thorough investigation including extensive laboratory tests, researchers found ozone to be the major fading agent with nitrogen oxides also capable of causing fading, but to a lesser extent. The fading mechanism is unique and complex. It takes place as a result of the curing operation and involves the disperse dyes on the polyester fibers rather than the vat dyes on the cotton. During curing some disperse dyes partially migrate to the permanent press finish. The finish is a combination of reactant resins, catalysts, softeners, and nonionic wetting agents. Dyes migrate to the solubilizing agents (nonionic surfactants and softeners) in the finish and are then in a medium in which fading by air contaminants can easily occur. Softeners are especially good media for absorbing gases.

The choice of catalyst used in the finish plays an important role. Migration of dyes increases significantly when magnesium chloride is used as a catalyst rather than zinc nitrate. Magnesium chloride is capable of forming complexes with certain anthraquinone disperse dyes (blues and reds), and these complexes are soluble in the resin finish. Therefore, to essentially eliminate this fading problem, textile processors must carefully select materials that make up the permanent press finish and/or replace vulnerable dyes with those that resist migration (36).

Fading of nylon carpets originated mainly in the warm humid areas from Texas to Florida, and, as a result, became known as "Gulf Coast Fading" (34,35). In one case, nylon carpeting in a Texas apartment complex showed visible fading 30 days after installation. A few incidents were also noted along the East Coast and in the Los Angeles area. Fading occurred on carpets manufactured from both nylon 66 and nylon 6 fibers and dyed with disperse dyes, which were used because many possess easy leveling properties necessary to avoid dye streaks. Fading took place largely on those carpets dyed with Disperse Blue 3 as a color component. Avocado, a tertiary-dyed dull-green shade, was a particularly sensitive color, and fading was characterized primarily by loss in blue as the green color gradually turned to a dull-orange shade.

Investigators eliminated sunlight as a primary cause of fading since fading occurred in rooms where light intensity was low. Exposure of carpet samples to standard test methods for ozone, also failed to duplicate the color change. Since fading occurred in humid environments and previous light-fastness testing and service trials established that conditions of high relative humidity promote fading of certain dyes by ozone, the investigators next exposed carpet samples to ozone in the presence of high relative humidity (85 to 90 percent). Under these conditions, pronounced fading took place on those samples containing Disperse Blue 3, and the fading was similar to the color changes observed in homes along the Gulf Coast.

Later experiments showed that relative humidity must be somewhat above 65 percent for pronounced ozone fading to take place. The fading problem may be prevented by using dyes that are more ozone-resistant and by using nylon fibers that have been modified (by dry-heat texturing) to decrease the accessibility and diffusion rate of ozone.

Recent research has also shown that the combination of ozone and high humidity causes pronounced fading of certain dyes on cotton and rayon fabrics. Consumers have complained about faded drapery and upholstery fabrics (35).

The United States Environmental Protection Agency has investigated the effects of air pollution on various dyed textiles by conducting both field and laboratory exposures. The field study consisted of exposing a wide range of dyed fabrics in light-tight cabinets located at four urban and corresponding rural (control) sites (37). These sites represented a cross section of various types of pollution and climates. The study was carried out over a 2-year period, with eight consecutive 3-month seasonal exposures.

Color change data, along with air pollution and weather measurements, were statistically analyzed in an effort to identify factors causing fading. The researchers found that about two-thirds of the fabrics showed appreciable fading. Most of these fabrics faded significantly more at urban sites than at corresponding rural sites, and the amount of fading varied between metropolitan areas and among seasons. Air pollution was assumed to account for most of the environmental differences between urban and corresponding rural sites. Analysis suggested that of the pollutants measured, NO_2, O_3 and SO_2 appeared to be those most responsible for fading. Generally, it was impossible to separate the effects of individual pollutants because they were confounded with the effects of other pollutants.

The controlled-environment laboratory study (38) was designed to assess the effects of common air pollutants, temperature, and relative humidity on the colorfastness of certain dyed fabrics selected from those exposed during the field study. Fabric samples were exposed to two concentrations of ozone: 100 $\mu g/m^3$ (0.05 ppm) and 980 $\mu g/m^3$ (0.50 ppm). As would be expected, high ozone levels produced significant fading, under similar exposure conditions, in more fabric samples than low levels, and furthermore, the color changes were more pronounced. Nevertheless, low ozone levels produced visible fading in a number of sensitive fabrics. This is important since the low levels were similar to levels frequently found in metropolitan areas. The study also clearly demonstrated that high relative humidity (90 percent), and to a lesser extent high temperature (32°C or 90°F), are significant factors in promoting and accelerating ozone-induced fading. This confirms what investigators observed during previous service trials. Rate-of-fading curves did not follow any consistent pattern; they were obviously dependent on such factors as color and type of dye, fiber substrate, and environmental conditions.

F. PAINTS

Recent laboratory research and field studies (39) indicate that ozone damages paint. The degree of damage depends on the type of paint. Although a number of techniques were used to assess damage, erosion measurements (weight loss converted to thickness loss) proved to be the most meaningful. Erosion, the gradual weathering of the surface of a paint film, is the normal mechanism of failure that limits the service life of a well-formulated and properly applied exterior coating.

The researchers selected five exterior coatings from four commercially important paint types: house paints - oil and acrylic latex; coil coating finishes - urea-alkyd; automotive refinishes - nitrocellulose/acrylic; and industrial maintenance coatings - alkyd.

Panels coated with the different exterior paints were exposed to controlled-environment conditions including clean air (control), clean air containing 196 $\mu g/m^3$ (0.1 ppm) ozone, and clean air containing 1960 $\mu g/m^3$ (1.0 ppm) ozone. The exposure chamber operated on a 2-hour light-dew cycle consisting of 1 hour of xenon light at 70 percent relative humidity and a black panel temperature of 66°C (151°F), followed by 1 hour of darkness at 100 percent relative humidity and 49°C (120°F) during which moisture condensed on the coated panels. One group of panels was exposed to simulated sunlight while a like group of panels was shaded during the exposures.

Erosion measurements were made after exposure periods of 0, 400, 700, and 1000 hours. Erosion rates for each paint type and exposure condition were then calculated from the erosion results. Zero-hour results, however, were excluded because erosion rates during the 0- to 400-hour period were greater than during the remaining exposure (400 to 1000 hours). The researchers concluded that this was primarily caused by removal of water-soluble materials and that materials subsequently eroded consisted mainly of binder and pigment.

Generally, exposures of 1960 $\mu g/m^3$ (1 ppm) ozone produced statistically significant increases in erosion rate compared to clean air (zero pollution) conditions. Erosion rate increases, however, varied considerably among paint types. Oil-based house paint experienced the largest erosion rate increase, industrial maintenance paint a moderate increase, and latex and coil coatings and automotive refinish the smallest increases. As would be expected, unshaded panels eroded more (degradation by sunlight) than shaded panels. Table 5-5 presents data for the unshaded exposures. Exposures to 196 $\mu g/m^3$ (0.1 ppm) ozone generally did not produce statistically significant erosion rate increases over clean air exposures.

One must bear in mind that erosion rate increases are differences between polluted and clean air exposures and therefore measure the influence of pollution; they do not necessarily imply overall durability. For example, in clean air unshaded panels of automotive refinish and coil coating experienced erosion rates of 0.5 and 3.0 nm/hr loss, respectively; however, erosion rates for both coatings increased only a small amount when they were exposed to high ozone conditions.

The researchers point out that in judging erosion rate increases, a distinction should be made between statistical and practical significance. For example, unshaded panels of automotive refinish developed an erosion rate of 0.5 nm/hr loss in clean air and 1.3 nm/hr loss in 1960 $\mu g/m^3$ (1 ppm) ozone. Both values are small and, although the increase is statistically significant, for practical considerations, the increase is not significant, especially in view of the severe

Table 5-5. EROSION RATES OF PAINTS EXPOSED TO LABORATORY-CONTROLLED OZONE ENVIRONMENTS

Paint type	Mean erosion rate (nm/hr loss) with 95 percent confidence limits for unshaded panels		Probability that difference exists, percent
	Clean air (control)	Ozone 1960 μg/m^3 (1.0 ppm)	
House paint - oil	5.1 ± 1.8	11.4 ± 2.7	99
House paint - latex	0.9 ± 0.4	2.2 ± 1.5	93
Coil coating	3.0 ± 0.6	3.8 ± 0.6	94
Automotive refinish	0.5 ± 0.2	1.3 ± 0.3	99
Industrial maintenance	4.7 ± 1.3	7.1 ± 3.6	85

exposure conditions. A similar analysis of oil-based paint erosion data, however, offers a reasonable degree of practical significance.

Field exposures were conducted in the following environments: clean rural, low sulfur dioxide (20 μg/m^3 annual mean), moderage sulfur dioxide (60 μg/m^3 annual mean) and high oxidant (40 μg/m^3 annual mean). Panels of the five different coatings were mounted at an angle of 85° from the horizontal to represent exposure conditions of normal house siding. Half of the panels were mounted facing south and half facing north. Panels were evaluated after 0, 3, 7, and 14 months' exposure. As with the laboratory data, the 0-month results were not used in calculating erosion rates.

Table 5-6 shows the erosion rate results for the various coatings exposed facing south. In most cases, southern exposures produced somewhat larger erosion rates, which agrees with the unshaded versus shaded results of the laboratory study. Oil-based house paint again had, by far, the largest erosion rate increases, followed in order by the coil coating, latex house paint, industrial maintenance paint, and automotive refinishes.

As in most field studies, it is difficult to designate air pollution or specific pollutants as significant causes of damage, especially when climatic conditions vary so widely. The high ozone and nitrogen oxide levels of the high oxidant site, nevertheless, suggest that one or both of these pollutants contributed to erosion rate increases measured at that site.

G. STEEL CORROSION

Haynie and Upham (40) found an interesting relationship between steel corrosion rates and ambient levels of sulfur dioxide and oxidants (mostly ozone). They statistically analyzed the results of a 5-year atmospheric corrosion study in which three different types of steels were exposed at urban and rural sites in eight metropolitan areas. All major air pollutants were continuously measured at or near each urban exposure site; no pollutants were measured at the rural control sites. The investigators found that more than 90 percent of the variability in corrosion behavior of the three types of steel exposed at urban sites can be accounted for by the variability in concentrations of two pollutants: sulfur dioxide increases corrosion and oxidants inhibit corrosion. Equations were developed for each steel that

Table 5-6. EROSION RATE OF PAINTS UNDER FIELD EXPOSURE

Paint type	Erosion rate (μm/mo) with 95 percent confidence limits and probability[a] for panels exposed facing south						
	Clean rural	Low SO_2 exposure (20 μg/m^3)	Prob-ability	Moderate SO_2 exposure (60 μg/m^3)	Prob-ability	High oxidant exposure (40 μg/m^3)	Prob-ability
House paint - oil	0.11 ± 0.19	0.38 ± 0.07	99	0.36 ± 0.12	98	0.53 ± 0.16	99
House paint - latex	0.05 ± 0.01	0.08 ± 0.02	99	0.10 ± 0.01	98	0.17 ± 0.14	94
Coil coating	0.05 ± 0.02	0.25 ± 0.05	99	0.24 ± 0.02	99	0.22 ± 0.04	99
Automotive refinish	0.02 ± 0.03	0.06 ± 0.02	98	0.04 ± 0.01	86	0.04 ± 0.01	92
Industrial maintenance	0.09 ± 0.04	0.21 ± 0.11	97	0.17 ± 0.10	91	0.20 ± 0.06	99

[a] Percent probability that differences exist compared to rural exposure.

related depth of corrosion to exposure time, mean sulfur dioxide concentration, and mean oxidant concentration. Relative humidity was not a factor because the range of average relative humidities between cities is small and rust remains moist most of the time at these relative humidities. Figure 5-1 shows the predicted effects of the two pollutants on the corrosion behavior of carbon steel exposed for 10 years. It should be noted that Figure 5-1 is an extrapolation of a nonlinear regression based on 5 years of data. Also, it is not

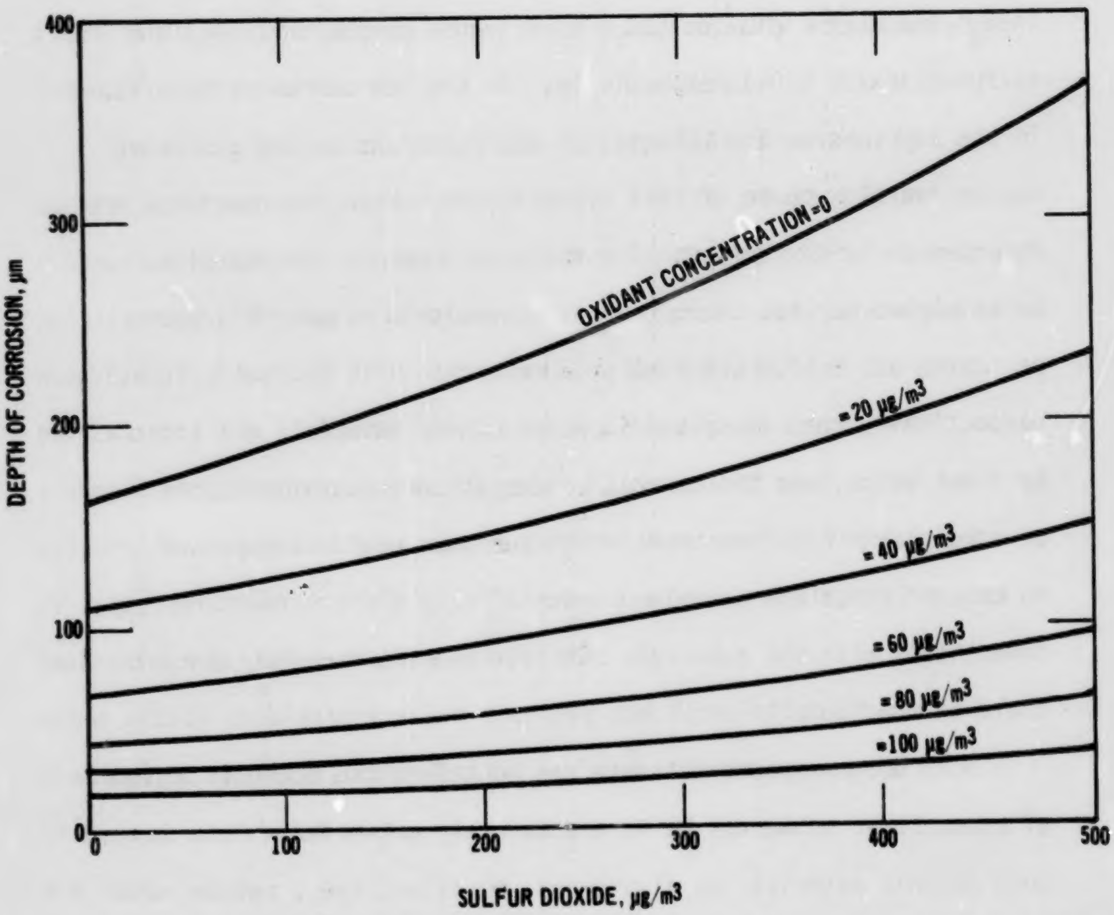

Figure 5-1. Effects of sulfur dioxide and oxidant concentrations on depth of corrosion of carbon steel exposed for ten years.

typical of carbon steels because this sample contained an unusually high concentration of copper, which improves corrosion resistance. Typical carbon steels, therefore, would be expected to be more susceptible to corrosion.

H. FUTURE RESEARCH NEEDS

Present knowledge of ozone damage to materials may be only a small fraction of the whole picture. The effects of interactions between ozone and other pollutants on materials has not been investigated. Based on thermodynamics, ozone should oxidize both nitrogen tetroxide (N_2O_4) and sulfur dioxide (SO_2) to nitrogen pentoxide (N_2O_5) and sulfur trioxide (SO_3) respectively. At the low concentrations found in the atmosphere, the kinetics of the reactions in the gas phase may be too slow to be of real significance. When the reactants are absorbed on solid surfaces of materials, however, the reactions may be catalyzed so that they occur at appreciable rates. Nitrogen pentoxide and sulfur trioxide produce nitric acid and sulfuric acid, respectively, when dissolved in water. Many materials are attacked by these acids, and the diurnal condensation-evaporation cycle of atmospheric moisture on material surfaces would be expected to concentrate these dissolved acids if they di[illegible]act immediately with the material. In this manner, damaging concentrations could be reached.

More dose-response data are needed before the economic impact of atmospheric ozone can be more accurately estimated. Data for such organic materials as elastomers, textiles, dyes, paints, and plastics are generally lacking. Nor have researchers conducted studies to determine dose-response relationships between multiple pollutants and various materials.

I. SUMMARY

Although the total extent of ozone-associated damage to materials is not known, ozone may very well be a major contributor to the weathering of materials. Ozone is an extremely active compound, and generally any organic material is susceptible to attack by concentrated ozone. Many organic polymers are subject to chemical alteration when exposed to very small concentrations of ozone, including atmospheric concentrations. This sensitivity usually increases with the number of double bonds in the chemical structure of the polymer.

The most widely used generic groups of elastomers are highly sensitive to attack by atmospheric ozone. These groups include natural rubber and synthetic polymers of styrene-butadiene, polybutadiene, and polyisoprene. Atmospheric ozone will not attack vulnerable elastomers when they are exposed in a relaxed state (no stress) even for long periods of time. However, cracks easily develop on exposure to atmospheres containing 20 to 40 $\mu g/m^3$ (0.01 to 0.02 ppm) ozone if vulnerable elastomers are under a tensile stress of as little as 2 or 3 percent. Unfortunately, elastomeric products are normally used in a stressed state. Other factors that determine the rate of ozone attack on elastomers are the type and formulation of the elastomeric material, concentration of atmospheric ozone, period of exposure, rate of diffusion of ozone to the elastomer surface, and temperature.

Researchers have developed antiozonant additives that are capable of protecting elastomers from ozone attack. Antiozonants, however, are expensive and sometimes migrate to the surface of elastomeric products where they may be lost during use. Oils, gasoline, and other chemicals may extract antiozonants from elastomers and thus decrease the resistance to ozone attack.

Researchers have conducted laboratory studies in which textiles have been exposed to ozone. Apparently only cotton is affected. Considering the above-normal exposure concentrations and the limited end-use life of most cotton products, the magnitude of deterioration by ozone is minimal.

Some important textile dyes, however, are susceptible to fading during exposure to ozone. Certain blue disperse dyes used on acetate and polyester/cotton fabrics, and on nylon carpets have been particularly troublesome. Laboratory exposures have shown that ozone concentrations as low as 100 $\mu g/m^3$ (0.05 ppm) are capable of producing visible fading in these materials. To prevent or mitigate fading by ozone, the textile industry can select combinations of fabrics, resistant dyes, and inhibitors, but these increase the cost of finished products.

Laboratory exposures of different types of paint have shown some damage at ozone concentrations of 1960 $\mu g/m^3$ (1.0 ppm), but essentially no statistically detectable damage at concentrations of 196 $\mu g/m^3$ (0.1 ppm). Oil-based house paint was most susceptible. Field exposures in Los Angeles (which has high smog pollution) also produced considerably more damage to some paints especially the oil-based house paint, than was observed at a rural (control) exposure site.

A recent atmospheric corrosion study indicates that the presence of oxidants (mainly ozone) inhibits the corrosion rates of several types of common steels. Increased oxidant concentrations correlate with decreased corrosion rates, even when atmospheric sulfur dioxide is present.

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CHAPTER 6

TOXICOLOGIC EFFECTS

A. INTRODUCTION

The photochemical process produces a number of irritating or toxic chemicals. This chapter discusses those, with an emphasis on ozone, which are likely to be toxic [illegible] near concentrations found in polluted ambient air. Also included are other secondary pollutants that might be implicated in eye irritation and the potentially toxic reactive hydrocarbons that, along with other factors, play a role in oxidant production. Photochemical oxidants such as ozone, nitrogen dioxide, and peroxyacyl nitrates are gases that exert their toxic influence through exposure via inhalation. (Nitrogen dioxide is reviewed in NATO/CCMS document N. 15). If present in sufficient concentrations, these gases are capable of causing death. At sub-lethal concentrations they produce more occult but nonetheless health-impairing physiologic malfunctions or anatomic lesions.

Data from exposure of any of the above agents at several orders of magnitude above the highest ambient concentrations, while of great basic toxicologic importance, are not relevant for ambient air. In this chapter, therefore, such information is included in tabular form to provide historical background and to develop a framework of relative toxicity for these compounds where clear-cut effects such as mortality, pulmonary edema, and the like can serve as end points. It is obviously impossible to detect such effects in community health surveys. It is likewise unwise to apply inferences drawn from animal exposure data at such concentrations directly as air quality criteria, since the mode of action of any given pollutant may be different at high and low levels of exposure. Where appropriate, however, experiments conducted at high concentrations will be cited for purposes of elucidating mechanisms, sites of action, or pertinent new information. For purposes of brevity, general high levels of toxicologic exposure data will be summarized in tabular form with an arbitrary cut-off for ozone at 1960 $\mu g/m^3$ (1 ppm) for 3 hours. The reader is referred to review articles by Stokinger (1), Jaffe (2), and Stokinger and Coffin (3) for additional discussion of this topic.

B. PATHOLOGICAL EFFECTS

Investigations of the acute toxicity of ozone (O_3) begun as early as 1904 determined that O_3 was a remarkably toxic gas. Early work was concerned mainly with mortality, gross examinations of the lung for evidence of inflammation, wet weight-dry weight ratios for the presence of edema and histopathological examinations, and the like. This work is summarized in Table 6-1.

1. Short-Term Exposures

The effect of exposure to 1960 $\mu g/m^3$ (1 ppm) O_3 and greater on eliciting acute pulmonary inflammation and edema has been studied. Applications of parameters of sufficient sensitivity have indicated that such changes may be detected at even lower concentrations.

Brief exposure to ozone elicits edema and an acute inflammatory response. Although it is striking at higher levels of exposure, the edemagenic response diminishes with descending concentrations of the gas. The exact threshold for this effect depends on the species of

Table 6-1. SUMMARY OF THE EFFECTS OF HIGHER CONCENTRATIONS OF OZONE ON ANIMALS

Ozone,[a] $\mu g/m^3$	Ozone,[a] ppm	Length of exposure	Observed effect(s)	Species	Reference
2,550	1.30	3 hours	Increased susceptibility to *Klebsiella pneumoniae*	Mice, hamsters	4
3,900	2.00	3 hours	Increased lung weight, decreased tidal volume, decreased minute ventilation	Rats	5
6,290	3.20	4 hours	Gross pulmonary edema	Mice	5
9,800	5.00	2 hours	Increased lung compliance, increased susceptibility to histamine	Guinea pigs	6
9,800	5.00	3 hours	Decreased activity of bacteriocidal lysozyme	Mice, rabbits	7
11,800	6.00	4 hours	Gross pulmonary edema, increased lung serotonin	Rats	8
20,000	15.00	30 min.	Decreased tidal volume, decreased O_2 consumption	Rabbits	5
41,000	21.00	3 hours	50% mortality	Mice	9
41,000	21.80	3 hours	50% mortality	Rats	9
67,980	34.50	3 hours	50% mortality	Cats	9
71,000	36.00	3 hours	50% mortality	Rabbits	9
101,370	51.70	3 hours	50% mortality	Guinea pigs	9
1,960	1.00	continuous	Bronchitis, bronchiolitis, emphysematous and fibrotic changes; acceleration of lung tumor development	Mice	10
15,700 to 88,000	8 to 45	1 hr/wk up to 49 wk	Damage to epithelium of the lower trachea and bronchioles; fibrosis	Rabbits	5

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[a] The concentrations of ozone listed are the lowest for which the observed effect has been recorded.

Table 6-1 (continued) SUMMARY OF THE EFFECTS OF HIGHER CONCENTRATIONS OF OZONE ON ANIMALS

Ozone,[a] μg/m^3	Ozone,[a] ppm	Length of exposure	Observed effect(s)	Species	Reference
			Systemic effects Short-term exposures		
1960	1.00	6 hours	60% increase in mortality as a result of exercise for 15 min/hr	Rats	1
6100	3.10	20 hours	Increased liver weight; increased liver alkaline phosphatase	Rats	11
7800	4.00	4 hours	Decreased mortality with age: young 50% mortality, old 10% mortality	Mice	1
11800	6.00	4 hours	Decreased brain serotonin	Rats	12
			Long-term exposures		

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[a] The concentrations of ozone listed are the lowest for which the observed effect has been recorded.

animals and the method employed to determine excess pulmonary water. Thus, no evidence of edema was noted in mice exposed to 1960 $\mu g/m^3$ (1 ppm) O_3 for 4 hours, whereas exposure to this concentration for 20 hours resulted in very slight edema when gravimetric measurement of lung water was the method employed (1,5). These investigators (1,5) detected evidence of edema in rats after exposure to 3920 $\mu g/m^3$ (2 ppm) O_3 for 3 hours. Exposure at the high level of 6272 $\mu g/m^3$ (3.2 ppm) O_3 quickly produced gross evidence of edema in mice. This phenomenon has been thoroughly studied in many species (Table 6-1).

While it is generally believed that a gross response by edema is probably not elicited in any species exposed to ambient concentrations of ozone, more refined methods for the detection of edema based on the recovery of radiolabeled blood albumin by means of pulmonary lavage, conducted by Alpert et al. (13), indicated that the ozone threshold for this phenomenon for rats lay between 490 and 980 $\mu g/m^3$ (0.25 and 0.50 ppm) O_3 for 6 hours.

Acute pulmonary inflammatory response is a regular feature of ozone exposure. Excess polymorphonuclear leukocytes were observed in pulmonary capillaries after exposure of mice to 1960 $\mu g/m^3$ (1 ppm) O_3 for 4 hours and inflammatory exudate was noted in the alveolar spaces after exposure to 1960 $\mu g/m^3$ (1 ppm) O_3 for 20 hours (5). The chemotactic effect of exposure to ozone has been shown at considerably lower concentrations and exposure times by methods employing pulmonary lavage and differential counts of the lavaged cells as studied by Coffin et al. (14,15). Thus, in rabbits a statistically significant increase in polymorphonuclear leukocytes removed from the bronchial tree through lavage was observed after exposure to 1960 $\mu g/m^3$ (1 ppm) O_3 and above for three hours. This response was noted immediately, peaked at about 6 hours after cessation of the exposure, and then gradually declined but was still evident 24 hours after exposure.

Scheel et al. (5), showed, by means of paraffin section, that exposure to from 1960 to 6272 $\mu g/m^3$ (1 to 3.2 ppm) O_3 for 4 hours in mice produced engorged capillaries containing an excess of polymorphonuclear leukocytes and desquamation of the superficial epithelial cells. Exposure at the higher level produced edema. Subsequent pathologic studies by other investigators (16) employing plastic embedded 1 µm sections as well as paraffin sections for light microscopy, and using transmission and scanning electron microscopy have further elucidated the pulmonary lesions of ozone. When Boatman and Frank (17) exposed cats to 510, 980, and 1960 $\mu g/m^3$ (0.26, 0.5, and 1 ppm) O_3 for 4 to 6 hours, lesions consisting of loss of ciliated cells throughout the airway were noted at all concentrations employed and appeared to be roughly dose-related. The most consistent ultrastructural changes in the airway were cytoplasmic vacuolizations of the ciliated cells and appearance of condensed mitochondria having abnormal cristae configuration. These changes appeared most pronounced in airways 0.8 to 1.7 mm in diameter. These same authors reported lesions consisting of swelling and desquamation of Type I alveolar lining cells, swelling and breakage of the capillary endothelium, and red cell lysis within the inter-alveolar capillaries. Similar findings from exposure to nitrogen dioxide (NO_2) had been noted, and the model system worked out previously by Freeman and coworkers (18-20).

Frank et al. (21) have reported essentially similar findings in rabbits exposed for 3 hours to 980 and 1960 μg/m^3 (0.5 and 1.0 ppm) O_3 in a model designed to elucidate the tolerance mechanisms in which one lung was collapsed by means of unilaterial (left) pneumothorax. For the purpose of this discussion of pathologic alterations, however, only the lesions induced in the right or non-tolerant lung will be described (see Section I. for additional information). These investigators report that with exposure to the higher concentration (1960 μg/m^3 or 1 ppm) O_3 up to one-third of the lining of the large airways was denuded of epithelial cells, with lesions in the small airways (0.3 to 0.17 mm in diameter) consisting of loss of cilia and vacuolization of the cytoplasm. At 980 μg/m^3 (0.5 ppm) O_3 changes were observed mainly in the larger airways. Abnormal mitochondria, increased electron density, and smaller longitudinally aligned cristae in the lower airway were noted at both concentrations. Changes noted in the alveoli by light microscopy consisted of focal areas of intra-alveolar hemorrhage and accumulation of leukocytes.

Study by means of transmission electron microscopy showed changes consisting of either swelling or desquamation of Type I cells and swollen or ruptured endothelial cells at the 1960 μg/m^3 (1.0 ppm) O_3 concentration with minimal swelling being apparent at the 980 μg/m^3 (0.5 ppm) O_3 level. Stephens et al. (22), studying the lesions induced by O_3 exposure in rats, have indicated that degenerative changes in the Type I alveolar cells occur after exposure to as low as 392 $\mu g/m^3$ (0.2 ppm) O_3 for 3 hours and that these cells are replaced by Type II alveolar cells beginning after one day. This same group of researchers has studied this lesion more extensively after NO_2 exposure and has worked out the sequences of destruction and desquamation and regeneration of alveolar cells. Presumably, these sequences are similar for ozone except that the lesion is induced by about 1/20th the dose for the more reactive ozone (18-20). Bils (23) investigated the effects of exposure of mice to 1176 and 2548 $\mu g/m^3$ (0.6 and 1.3 ppm) O_3 for 4 hours by means of electron microscopy and noted essentially similar effects from both concentrations, namely swelling of the epithelial alveolar lining cells but no evidence of fluid accumulation in the alveolar space. Exposure to these concentrations was also associated with focal swelling of the endothelium cells and an occasional break in the basement membrane.

Electron microscopic studies of lungs of animals exposed to ozone have also been carried out by Werthamer et al. (24) and Plopper et al. (16,25) at concentrations which are known to produce gross evidence of edema but which are several times higher than ambient concentrations. Since these data provide a continuum of effects at higher levels, however, they provide an upper dose response point. These studies, by virture of their stronger effect, characterize some of the lesions seen from exposures to ambient or near-ambient concentrations, thus providing a link with earlier studies made by light microscopy after exposure to lower concentrations of ozone.

Werthamer et al. (24) found acute inflammatory bronchiolar lesions following ozone exposure of mice to 7840 $\mu g/m^3$ (4 ppm) for 3 hours. Plopper et al. (16), reported findings from exposure of rats to approximately 5880 $\mu g/m^3$ (3 ppm) O_3 for 4 hours. Histopathological observations made by light microscopy on freeze-substituted sections indicated irregularly distributed focal or confluent lesions. Changes predominating were bronchiolar epithelial necrosis and sloughing, and alveolar edema, particularly in the region of the terminal airway units. These changes are essentially the same as those described by others using standard histologic methods to examine tissues from animals exposed to high levels of ozone.

Ultrastructural studies on gluteraldehyde-fixed sections embedded in epoxy indicated that cilia and epithelial surface projections were diminished on the bronchiolar epithelial cells (20). The same changes had been previously noted for NO_2 by Freeman et al. (18). In addition to edema and fibrillar exudative elements in the alveoli, specific cellular injury was observed. Swelling of the membranous pneumocyte (Type I alveolar cells) was noted. Additionally, there were cytoplasmic swelling and internal blebing of the capillary endothelium. Interstitial edema was also present. The cellular lesions appeared to be confined to the proximal portion of the pulmonary acini and adjacent terminal bronchioles. Endothelial lesions, however, were observed peripherally as well as proximally in the acini.

2. Effects of Longer Exposure

A number of investigators have either continuously or intermittently exposed various animals to ozone over considerable periods. Stokinger et al. (10) reported that chronic bronchitic, bronchiolitic, emphysematous, and fibrotic changes occurred in the lungs of rats, mice, hamsters, and guinea pigs exposed 6 hours daily, 5 days a week for 14 months to 1960 $\mu g/m^3$ (1 ppm) O_3 Freeman et al. (26) and Stephens et al. (27) have reported on light and electron microscopic studies of the lungs of dogs exposed to doses ranging from 1960 to 5880 $\mu g/m^3$ (1 to 3 ppm) O_3 for 8 to 24 hours daily for a period of 18 months. The various changes observed appeared roughly proportional to the time and dose of the ozone exposure. These consisted of increase in the number of pulmonary alveolar macrophages beginning at exposures of 1960 $\mu g/m^3$ (1 ppm) O_3 for 8 hours daily; deposition of fibrous elements; and thickening of the terminal airway and respiratory bronchiolar walls with consequent reduction of the lumina of the small airways. There was also an accumulation of lymphocytes, plasma cells, and fibroblasts in the peribronchiolar tissue, with an increase in the proportion of mucus-secreting cells and squamous metaplasia of columnar and cuboidal cells.

Ultramicroscopic studies performed on dogs exposed to 5880 μg/m^3 (3 ppm) O_3 for 8 hours per day for 18 months indicated abnormalities in the granular pneumocyte (Type II alveolar epithelium) and in the endothelial cells of the interalveolar capillaries. The investigators speculated that these changes were caused by metabolic alterations of the cells. These changes consisted of an accumulation of a material with a recurring periodicity of 754 Å within dilated cisternae of the rough endoplasmic reticulum (ER) or in large individual cisternae in the rough endoplasmic reticulum of the Type II cells. Ribosomes and rough ER were more abundant in Type II cells. Accumulation of tubular membrane complexes forming lattice work-like structures in the cytoplasm of capillary endothelial cells was also found. The significance of the changes is not clear.

Freeman et al. (28) have studied rats exposed to 1058 and 1725 μg/m^3 (0.54 and 0.88 ppm) O_3 for varying periods up to 6 months. They report the pathological sequence from observations in lungs perfused with Zenkers fluid at a pressure of 25 cm of water. Tissue blocks were embedded in paraffin and sectioned at 4 and 16 μm. Observations made on animals exposed to 1058 μg/m^3 (0.54 ppm) O_3 and sacrificed after 12 hours of exposure were essentially negative. After 24 hours, however, the respiratory bronchioles and alveolar ducts contained hypertrophic epithelium as well as amorphous material, suggesting the presence of necrotic cells adjacent to the terminal bronchioles. After 48 hours, the alveolar ducts had a hypertrophic alveolar epithelium that contrasted sharply with controls. Numerous mitotic figures were present in the terminal bronchiolar and proximal alveolar epithelium. In the proximal air spaces, macrophages were prominent, and there was indication that fibroblasts were increased in the sub-epithelial tissue of the respiratory bronchioles in some places amounting to replacement of the respiratory epithelium. Additional animals were sacrificed at 6 days, 8 days, 3 weeks, and 6 months. Progressive changes in the epithelial cells were reported. The hyperplastic epithelium became cuboidal and was replaced more and more by connective tissue. There was a gradual reversion of the connective tissue to a less cellular appearance in the terminal bronchioles. Connective tissue deposits, however, tended to disrupt the symmetry of the small airways and proximal alveoli, and possibly restrict the lumina of the fine airways.

When rats were exposed to 1725 $\mu g/m^3$ (0.88 ppm) O_3 evidence of epithelial injury was present as early as 4 hours after beginning the exposure. In general, the observations noted during the 1058 $\mu g/m^3$ (0.54 ppm) O_3 exposure were exaggerated and occurred sooner. Additionally, after 2 days of exposure at the higher concentrations, metaplastic-like activity associated with fibrosis was noted in the bronchiolar epithelium. As the lesion progressed, replacement of normal alveolar epithelium by cuboidal cells (bronchiolization of the alveoli) was noted, as were the presence of adenoma-like structures, abundant macrophages, invasion of fibroblasts, and flattening and smoothing of the bronchiolar epithelium. After exposure of 3 months to the higher concentrations, full-blown emphysema-like lesions were observed. The animals exhibited gross enlargement of the thorax, strong kyphosis, and grossly enlarged lungs which retained more air when removed from the chest cavity. Additionally, there were fine structural changes consisting of enlargement of the pulmonary air spaces as well as cellular changes most prominent in the central area of the pulmonary acini discussed above. These appear nearly identical to those reported for exposure to nitrogen dioxide at higher concentrations and for longer periods.

C. BIOCHEMISTRY

The biochemical mechanisms by which exposure to ozone damages biological systems has been the subject of considerable speculation and investigation. Among the mechanistic postulations which have been advanced are the following: 1) Non-specific stress with the release of histamine, 2) Oxidation of the sulfur-bearing compounds (sulfhydryl groups) or their precursers, 3) Oxidation of polyunsaturated lipid contained mainly in cell membranes, 4) Formation of toxic compounds (ozonides) through reaction with polyunsaturated lipid, 5) Formation of free radicals, 6) Injury mediated by a pharmacologic action, i.e. , via a neurohormonal mechanism.

Despite rather extensive investigation of the problem, there is still no generally accepted explanation of the biochemical mechanism or mechanisms through which ozone exerts its toxic action.

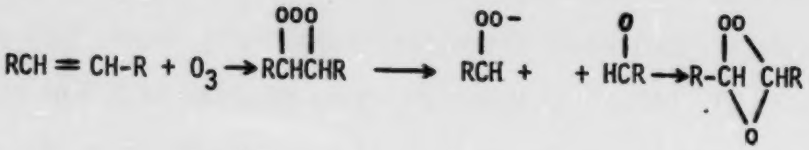
Dixon and Mountain (29) have shown that the release of histamine from the lung occurs immediately after exposure to ozone and persists for as long as 11 days in mice and 17 days in rats. Pre-treatment with the antihistamine (promethazine) reduced pulmonary edema following an edemagenic dose of ozone. The effects of aspirin in reducing the effects of ozone-induced edema may be related to its recently demonstrated ability to block prostaglandin synthetase. Bradykinin appears to be intimately related with prostaglandins in the lung and potentiation is possible.

Fairchild et al. (30) suggested that edema was inhibited by the prior administration of agents which blocked the histamine-sensitizing factor. Easton and Murphy (6) demonstrated that ozone-exposed animals reacted more severely to parenterally administered histamine.

Oxidation of the sulfhydryl groups (SH) was indicated by Fairchild et al. (31) in 1959. They showed that the addition of SH compounds was protective. Subsequent studies by a number of investigators have confirmed these findings and extended the depth of study. Mudd et al. (32) studied ozone oxidation of glutathione in an in vitro model and showed that both fast and slow oxidation occurred. Studies by Mountain (33) showed that in vivo ozone exposure caused oxidation of reduced glutathione (GSH) and depression of succinate dehydrogenase activity in the lung. Studies by King (34) demonstrated the loss of SH content and loss of enzymatic function of partially purified glyceraldehyde-3-phosphate dehydrogenase in rat lungs following exposure to 2352 $\mu g/m^3$ (1.2 ppm) O_3 for 4 weeks. Menzel (35) and Mudd (32) demonstrated that the oxidation of GSH proceeded to oxidized glutathione (GSSG) though some of the -SH groups formed higher oxidation products (i.e., sulfoxide and sulfone). DeLucia et al. (36) found that the concentration of protein sulfhydryls (PSH) and non-protein sulfhydryls (NPSH) were reduced in the lungs of rats exposed to 3920 $\mu g/m^3$ (2 ppm) O_3 for 4 to 8 hours. Additionally, several lung enzymes, glucose-6-phosphate dehydrogenase, glutathione reductase, and NADH- and succinate-cytochrome C reductase were reduced by similar exposure. DeLucia et al. (36) have also reported that exposure to as little as 392 $\mu g/m3$ (0.2 ppm) O_3 for 4 hours results in a statistically significant depression of GSH and associated enzymes up to 24 hours after exposure followed by a subsequent elevation above normal, that persists for several days. It is also well known that supplementing with glutathione or its precurser, methionine, blocks this effect and goes far in mediating the various other manifestations of ozone exposure (37,38). It should be pointed out, however, that the cycle of depression and augmentation of lung glutathione mimics the cytological cycle induced by ozone exposure, namely necrosis of Type I alveolar epithelial cells with subsequent cellular hyperplasia, particularly of Type II cells said to be rich in these compounds. Thus, it is possible that the GSH reduction cycle is a result, rather than a cause, of ozone injury. Systemic reduction of reduced glutathione cannot be overlooked since blood GSH levels were suppressed throughout exposure to 1960 $\mu g/m^3$ (1.0 ppm) O_3 for up to 60 days.

Oxidation (peroxidation) of polyunsaturated fatty acids has been advanced as a toxic mechanism in both ozone and nitrogen dioxide injury by Thomas, Mueller, and Lyman (39), Goldstein et al. (40), and Roehm et al. (41). This postulation is based on the proclivity of ozone to react with the ethylene groups of the unsaturated fatty acids. This reaction can lead to free radicals, which in the presence of molecular oxygen leads to the peroxidation of the unsaturated fatty acid by formation of fatty acid lipoperoxides. In support of the hypothesis that a factor in ozone toxicity is peroxidation of polyunsaturated fatty acids, Goldstein et al. (42) and Menzel et al. (43) have performed experiments which indicate that a deficiency of Vitamin E, the principal cellular free radical trap, increases the toxicity of ozone in the rat. Goldstein et al. (40) speculate that this process could be effected either by direct ozonolysis and perpetuated by from unsaturated fatty acid breakdown. If such proved to be the case, a chain reaction beginning with direct ozonlysis and perpetuated by the resultant free radicals could be envisioned as a consequence of ozone exposure.

Another chemical pathway derived from unsaturated fatty acid oxidation by ozone has been demonstrated and might be considered a mode of toxicity. Roehm et al. (41) studied the oxidation of polyunsaturated lipid in an in vitro model in which samples of pure methyl esters of fatty acids were exposed to dynamically flowing atmospheres of ozone and nitrogen dioxide. There was a marked difference in the mode of oxidation between the two gases, ozone being the most reactive. Oxidation by ozone was not completely inhibited by phenolic antioxidants. Methyl linoleate was completely reacted by ozone to yield a number of compounds including monozonides and diozonides. The authors speculate that ozone acts by direct addition of O_3 across the fatty acid double bond, postulating that the mechanism proposed by Criegee (44), is the primary mechanism for ozonolysis. This reaction proceeds as follows:



$$RCH = CH\text{-}R + O_3 \rightarrow \overset{OOO}{RCHCHR} \longrightarrow \overset{OO-}{RCH} + \quad + \overset{O}{HCR} \rightarrow R\text{-}\overset{OO}{CH}\ \underset{O}{\ }\ CHR$$

The ozonide, once formed, appears stable. Menzel et al. (43) have studied the effects of fatty acid ozonides *in vivo* and with *in vitro* models. Intradermal injections of fatty acid ozonides of oleic, linoleic, linolenic, and arachnidonic acids (10 picograms to 10 micrograms) elicited increased vascular permeability of the capillaries as measured by pontamine blue bound serum proteins. The reaction appeared to be mediated by histamine since it was blocked by prior treatment with compound 48/80 or by simultaneous injection by antihistamines. According to these same authors, fatty acid ozonides also rapidly initiated peroxidation of isolated microsomes and mitochondria. They also observed that erythrocytes incubated with fatty acid ozonides formed Heinz bodies and lysed, indicating oxidation of the red cell membrane and suggest that fatty acid ozonides may be the toxic intermediaries in ozone toxicity.

Evidence for the release of free radicals by ozone rests on pathologic observations, physical-chemical theory, and some actual observations. So-called radiomimetic properties have been ascribed to ozone on the basis that certain pathologic events are common to both ozone and ionizing radiation.

The pathologic alterations produced from ozone exposure which has been likened to those caused by ionizing radiation may be listed as follows: 1) Chromosome aberrations reported by Fetner (45) and by Zelak et al. (46). 2) Premature aging reported by Scheel et al. (5). 3) Retardation in the deoxygenation of hemoglobin in the capillary blood (common to radiant energy) reported by Brinkman and Lamberts (47). 4) Avoidance behavior in mice described by Peterson and Andrews (48). Theoretical support for the existence of free radicals are as follows: While ozone is predominantly an ion, it reacts to generate free radical species because of the vigorous nature of the oxidant and the formation of peroxy intermediaries. Free radical-mediated chain reactions can thus occur on oxidation of biological material. There is fairly good theoretical support that ozone reacts with sulfhydryl groups oxodizing them to thionyl free radical prior to oxidation to higher oxidation states (e.g., sulfoxide and sulfone). It also has been speculated that during the peroxidation of polyunsaturated lipid by ozone, free radicals are liberated via peroxy intermediaries. Other facts that may be indicative of free radicals from ozone are that protection is provided against both ozone and ionizing radiation by sulfur compounds which are known to be free radical scavengers as pointed out by Fairchild et al. (31). Breakdown products of oxidized lipid include substances that may cause polymerization and cross-linking of protein as suggested by Buell et al. (49). Finally, free radical signals have been demonstrated by electron paramagnetic resonance spectrometry during the ozone catalyzed oxidation of linoleic acid *in vitro* by Goldstein et al. (40).

Neurohumoral reaction to ozone exposure has been indicated in reports by Fairchild and co-workers (50-52). Evidence for the involvement of the pituitary-thyroid-adrenal axis stems from the fact that hypophysectomy, thyroidectomy, and adrenalectomy will increase the resistance of rats to otherwise lethal doses of ozone by decreasing the elicitation of pulmonary edema (50). Ablation of the thyroid has the strongest effect, followed with decreasing importance by the pituitary and adrenals. The administration of antithyroid drugs (such as methimozole, thiourea and the like) likewise increases resistance to the lethal effects of ozone exposure (50) while, conversely, hormonal preparations, such as thyroxin, increase the edemagenic response. Fairchild et al. (51) have further demonstrated that exposure to as little as 980 $\mu g/m^3$ (0.5 ppm) O_3 inhibits the release of I^{131} from the thyroid. It has been suggested that the inhibitory role of the endocrine gland ablation rests on the elaboration of pressor amines and consequent hemodynamic shifts, rather than alteration of metabolic rate since production of a similar effect has been reported from lesions of the spinal cord at the sixth and seventh cervical and second thoracic segments (52).

D. PHYSIOLOGIC ALTERATION

A number of studies have been conducted to determine the physiologic response of the lungs of several species of animals from exposure to ozone. Most of these studies suffer from the difficulties associated with performing delicate pulmonary mechanical maneuvers on the lungs of small non-cooperative animals, the necessity of employing anesthesia, and particularly, the use of unrealistically high concentrations of the gas, which may introduce artifacts into the data. Possibly, the most effective use of animals has been in developing models for gas absorption in the respiratory tract and evaluating various mechanisms of ozone action as they are reflected in animal models in which various surgical or pharmacologic interventions can be conveniently carried out. For determining the influence of ozone on the pulmonary physiologic parameters of man, the best model is man himself. Consequently, several investigators have carried out experimental exposures on human volunteers to low, but physiologically reactive, concentrations of ozone.

The respiratory uptake of ozone has been measured in dogs by Vaughan et al. (53) and Yokoyama and Frank (54) by means of surgically isolating the upper airway. In the first study, the uptake in the upper airway alone was 40 to 60 percent in dogs exposed to from 17,640 to 23,520 $\mu g/m^3$ (9 to 12 ppm) O_3 and 15 to 20 percent when the animals were exposed to concentrations of 13,720 $\mu g/m^3$ (7 ppm) O_3 or less. In the second study, where exposures were to concentrations of 392 to 784 $\mu g/m^3$ (0.2 to 0.4 ppm) and 1372 to 1666 $\mu g/m^3$ (0.7 to 0.85 ppm) O_3, the rate of uptake was greater at lower velocities and at lower concentrations. The rate of uptake was twice as high in the nose as in the mouth, but highest in the lower airway (80 to 87 percent). The investigators estimated that approximately 90 percent of the ozone in respired air was taken up during quiet respiration. It is speculated that mouth-breathing may be a factor that heightens the effect of exercise in human subjects. Work is being carried on utilizing this type of study to derive other data for the construction of a mathematical uptake model at various segmental levels of the upper and lower respiratory tract. Preliminary computations indicate that uptake is a function of gas solubility; thus with ozone, which is slowly soluble, penetration is deep. The preliminary model predicts that the peak uptake for ozone occurs between the 16th and the 20th segmental level of the human lung (55).

Physiologic response of the lungs has been studied by Murphy, et al. (56) in guinea pigs employing the forced pressure oscillatory method. Pulmonary flow resistance was not significantly altered by 666 and 1336 $\mu g/m^3$ (0.34 and 0.68 ppm) O_3, but increased 47 percent with exposure to 2117 and 2646 $\mu g/m^3$ (1.08 and 1.35 ppm). Watanabe et al. (57) studied the effect of ozone exposure on pulmonary mechanics in cats exposed to 510, 980, and 1960 $\mu g/m^3$ (0.26, 0.5, and 1 ppm) O_3

for 2 to 6.5 hours. Their findings indicate that pulmonary flow resistance was increased more or less proportionally to the ozone dose while dynamic compliance fell less frequently and with less amplitude. There was no change in vital capacity, but the frequency of reduction of the diffusing capacity of the lung among the various animals exposed (measured with carbon monoxide) appeared to be a function of ozone concentration and there was an apparent association of reduced diffusion capacity and increased airway resistance in individual animals. The authors point out that the change in airway resistance had reflex and nonreflex components. Concomitant pathological studies on similarly exposed animals showed severe injury to the bronchial and bronchiolar epithelium, alveolar Type I cells, and interalveolar capillaries, though no evidence of intra-alveolar edema was observed (see Section A).

Several investigators have studied the influence of exposure to ozone on extrapulmonary physiologic parameters. Exposure to ozone reduced the gross motor activity of guinea pigs, rats, and mice. Murphy et al. (56) found depression of spontaneous running activity of mice after exposure to 392 and 980 $\mu g/m^3$ (0.2 and 0.5 ppm) O_3 for 6 hours. Konigsberg and Bachman (58) also noted a diminution in gross motor activity of rats and noted it appeared related to concentration of ozone where exposures varied from 196 to 1960 $\mu g/m^3$ (0.1 to 1.0 ppm.)

The effect of vagotomy has been studied by Vaughan et al. (59) in dogs exposed to from 78,400 to 107,800 $\mu g/m^3$ (40 to 55 ppm) O_3

for 5 minutes. They produced an apneic phase of approximately 1 minute associated with a slight fall in blood pressure and slowing of the heart rate. These changes, which did not occur at the 78,400 $\mu g/m^3$ (40 ppm) O_3 level, appeared to be blocked by vagotomy. Dynamic compliance was reduced and pulmonary flow increased in both non-vagotomized and vagotomized animals.

The influence of unilateral exposure on pulmonary physiologic parameters has been studied by Frank et al. (60) and Alpert et al. (61). In the former study, unilateral exposure was accomplished by means of one-sided pneumothorax. The animals were subjected to a unilateral exposure either of 3724 or 10,192 $\mu g/m^3$ (1.9 or 5.2 ppm) O_3 for 3 hours to the right lung. Following these exposures, the pneumothorax was removed and the animals exposed bilaterally to 3724 or 10,192 $\mu g/m^3$ (1.9 or 5.2 ppm) for 3 hours. The wet/dry weight ratios were lower in the pre-exposed right lung than in the single exposed left lung. There was no evidence that the left lung was resistant to edema. The distensibility of the pre-exposed right lung as measured by its slow V/P behavior was lower than the paired, more edematous left lung. The second study (61) consisted of unilateral exposure via tracheotomy and unilateral bronchiolar catheterization in Flemish giant rabbits. The ozone dosage was 23,520 $\mu g/m^3$ (12 ppm) for 6 hours. The respiratory rate increased for about 3.5 hours, then gradually declined. Tidal volume fell in both control and O_3 treated lungs with the terminal values for the treated lungs falling below the control. No unilateral functional changes occurred prior to the development of edema. The authors speculate that changes might be due to neural or chemical response, possibly secondary to edema.

Changes in the elasticity of the lung have been reported from exposure of various animal species to ozone. Reduction in compliance has been reported by Alpert and Lewis (61) in rabbits, Vaughan et al. (59) in dogs, and Frank et al. (60) in rabbits. These exposures were all above the edema-producing level and quite likely are the result of the influence of edema fluid increasing the rigidity of the lung. Watanabe et al. (57), however, reported a slight irregular fall in compliance in cats exposed to concentrations of 1960 $\mu g/m^3$ (1 ppm) O_3 and less. Yokoyama (62) has also reported the reduction of variations in compliance in isolated dogs' lungs and a significant decrease in the pressure volume relationship after exposure to 9800 to 19,600 $\mu g/m^3$ (5 to 10 ppm) O_3. Perhaps the most natural exposure of the lung that caused significant alteration of the pressure volume relationship was carried out by Bartlett et al. (63) who exposed rats to 392 $\mu g/m^3$ (0.2 ppm) O_3 continuously for 30 days. Lung volumes were increased 16 percent and static pressure volume measurements indicated that the lungs were over-distended by high transpulmonary pressures. These changes were interpreted by the authors as indicating a decrease in lung elasticity and suggest mediation by an effect on collagen.

It is probably relevant to the subject of lung elasticity to discuss the role of ozone in alteration of the pulmonary surface-active material under pulmonary physiology since it is known that this substance provides stability to the pulmonary parenchyma by virtue of its reduction of surface tension when compressed. Mendenhall and

Stokinger (64) showed an attenuation of surface pressure when lung washings were exposed to ozone _in vitro_. This action could be blocked by the addition of SH-containing substances. Yokoyama (65) noted a questionable increase in minimal surface tension after _in vitro_ exposure of lung extracts to 980 to 2352 μg/m^3 (0.5 to 1.2 ppm) O_3. No change was evident when similar experiments were carried out with dipalmetoyl lecithin. However, no alterations in surface tension of either pulmonary lavage fluid preparations or of dipalmetoyl lethicin were noted by Gardner et al. (66). It is not possible theoretically for ozone to attack the highly saturated molecule of dipalmetoyl lecithin generally concluded to be the principal of this substance. Thus, if changes in surface pressure do occur, it is not by alteration of dipalmetoyl lecithins and some other mechanism must be postulated.

E. EXPOSURE OF HUMAN VOLUNTEERS

A number of studies of pulmonary physiologic response employing experimental exposure of human beings have been conducted. The subject has been recently reviewed by Bates and Hazuka (67). Griswold et al. (68) exposed one person to 3920 μg/m^3 (2 ppm) O_3 for 2 hours and noted a transient fall in vital capacity and forced expiratory volume. Clamann and Bancroft (69), in an exposure of approximately 1960 μg/m^3 (1 ppm) O_3 for 2 hours, saw reduction in vital capacity and maximal breathing capacity. Bennett (70) exposed subjects to 980 μg/m^3 (0.5 ppm) O_3 for 3 hours per day for 6 weeks and produced a progressive decrease in the one-second forced expiratory volume.

Lagerwerff (71), however, noted no change in vital capacity after a single exposure to 980 μg/m^3 (0.5 ppm) O_3 for 3 hours. More extensive exposures were conducted by Young et al. (72) on 11 volunteers via mouthpiece apparatus. Exposures were from 1176 to 1568 μg/m3 (0.6 to 0.8 ppm) O_3 for 2 hours. They reported a significant reduction in steady state pulmonary diffusing capacity and in forced respiratory volume in their subjects. Hallett (73) exposed volunteers in a study which proved retrospectively to be from 1960 μg/m^3 (1 ppm) to possibly as great as 5880 μg/m^3 (3 ppm) O_3. As might be expected, the data showed a variable response in vital capicity, forced expiratory volume, and diffusion capacity. Experiments were conducted by Goldsmith and Nadel (74) on four human volunteers by exposure to 196 to 1960 μg/m^3 (0.1 to 1.0 ppm) O_3 for one hour. The investigators concluded that there was an increase in airway resistance at concentrations of 1176 μg/m3 (0.6 ppm) O_3 and above.

A significant change in the regimen of exposure was undertaken by a group of investigators at McGill University who have evolved an experimental protocol containing a second variable, light exercise, in conjunction with exposure to ozone (67,75,76). The exercise was standardized so that the volunteers alternated 15 minutes of exercise on a bicycle ergomometer with 15 minutes of rest. The exercise employed was sufficient to double the minute ventilation and was stated by the authors to be equivalent to walking on a level surface. Exposure took place in a plexiglass enclosed space by natural respiration, thus providing a much more natural exposure than hitherto used for human volunteers where face masks, mouth pieces, and nose clips had been employed. In addition to the more routinely applied lung function tests, residual volume employing a helium dilution method and closing volume using a bolus of 133 xenon were performed (67,76). The importance of this latter technique will be discussed below.

Data from exposures employing intermittent exercise may be summarized as follows: significant decrement in maximal mid-expiratory flow rate at 725 and 1470 μg/m^3 (0.37 and 0.75 ppm); significant decrement in forced expiratory volume 725 and 1470 μg/m^3 (0.37 and 0.75 ppm) O_3. On the basis of these data, the authors have been able to calculate the probable effect of varying concentrations of ozone on forced vital capacity, forced expiratory volume, and the maximum mid-expiratory flow rate.

Based on recent pathological anatomical evidence, this same group has considered the possibility that the small airways likewise might be a major site of ozone action on pulmonary functional parameters. Since the customary tests used to measure ventilatory performance are insensitive to changes in the small airways, application of tests to measure closing volume as a percent of vital capacity, or closing capacity (the latter measuring the closing point as a percent of total lung capacity) were utilized. The latter index appeared to be more appropriate for the ozone experiments since vital capacity was a changing phenomenon during these experiments. Their data indicate that there was a considerable increase in closing capacity (inflection point had moved to the left). According to the investigators, this indicated that there were obvious effects in the small airways. The volunteers were categorized as smokers and non-smokers for the above experiments. Results indicate that while differences between smokers and non-smokers were questionable at the 725 $\mu g/m^3$ (0.37 ppm) concentration, exposure at 1470 $\mu g/m^3$ (0.75 ppm) O_3 produced more severe decrements in function parameters in smokers.

Human volunteers generally experience pain or discomfort while undergoing exposure. Subjective symptoms reported have included cough, substernal soreness, pharyngitis, dyspnea, headache, and conjunctivitis.

F. OZONE INTERACTION WITH MICROBIAL AGENTS

Environmental toxicology has generally been concerned with the association of single agents with effects. However, as both Henderson (77) and Topps (78) have pointed out, spontaneous diseases are usually the product of several factors operating in concert. This association of several intrinsic and extrinsic factors in disease production has been termed the multiple causality principle. Interaction of several air pollutant factors may synergistically increase effect. Equally possible is the biological interaction of a disease-producing microorganism and an air pollutant. These effects were seen by Stokinger et al. (79) who noted that exposure to both thiols and ozone appeared to activate bacterial infections of the lung at well below the concentrations of these gases which would in themselves be expressed in fatal toxicity. Similar effects were noted by Hueter et al. (80) in animals undergoing exposure to synthetically produced smog.

Studies have been instituted by several investigators to examine this phenomenon experimentally by exposure to various sequences of air pollutants and aerosolized bacteria or filterable viruses. Under such conditions, very effective models have been reported for ozone, nitrogen dioxide, and synthetic smog.

1. Ozone

The capacity of exposure to ozone to enhance mortality from bacterial organisms has been the subject of many reports. Miller and Ehrlich (4) noted enhancement of mortality to subsequent exposure to either Streptococcus or Klebsiella. This effect was found to persist from 6 and 19 hours when the challenge aerosol was presented after the exposure to ozone, and up to 27 hours when the pollutant exposure followed the bacterial aerosol. The model usually employed the sequence of exposures to the pollutant followed by the bacterial challenge (4,81,82). Subsequent studies by Coffin and Blommer (83,84,85), Coffin (86), and Coffin et al. (87) have established an effect threshold for ozone at 157 $\mu g/m^3$ (0.08 ppm) for 3 hours. The subject has recently been reviewed by Coffin and Gardner (15).

Exposure to a third interactant such as a biometeorological variable or exercise appear to augment the effect of ozone in enhancing mortality from infection. Coffin and Blommer (83) noted that exposure to 3° to 5°C for 4 hours prior to the ozone exposure and holding the animals at 35°C after the bacterial challege heightened gross mortality differences attributable to the ozone exposure. The use of forced exercise in mice is conducive to larger differences in the ozone-stimulated diminished susceptibility to bacterial infection (88). Similar effects from exercise have previously been noted by Stokinger (89) in terms of gross mortality.

2. Photochemical Smog

Experiments performed on pathogen-free random bred Swiss mice, in which exposure to synthetic smog was followed by bacterial challenge with streptococci, indicate that photochemical smog has the ability to potentiate infection (84). The photochemical smog was produced by subjecting diluted auto exhaust to fluorescent light to produce the experimental atmosphere (90). Enhancement of mortality over filtered air controls was noted at concentrations of 25 ppm CO and 0.15 ppm total oxidant, well within the levels commonly found in polluted atmospheres. The effective chemical was presumably the oxidant since CO is not known to be effective in this system and NO_2 present in approximately equal amounts to the O_3 is probably ineffective in short exposures at such concentrations.

3. Mechanisms of Action

Mechanisms by which air pollutants diminish the resistance of animals to air pollution have been studied for the oxidant gases, principally ozone. Such exposures alter the animals' defenses so that bacteria are killed more slowly in the lungs and grow at an accelerated rate irrespective of the number of organisms that remain after the original clearance period. It would appear that the oxidant pollutants remove whatever influences the inhibition of bacterial growth and permit the organisms to grow uncontrollably to produce disease rapidly. The result is a more rapid replication of organisms, earlier invasion of the blood, and earlier death in ozone-treated animals as compared to controls. Several references (15,82,85,86) treat this topic in greater detail.

Possible mechanisms for the action of ozone, nitrogen dioxide, and photochemical smog might be: 1) Greater deposition of organisms; 2) Edema of the lungs; 3) Effect on cellular components; 4) Effect on noncellular milieu; 5) Effect of anatomic lesions (91).

Deposition of organisms played no part in mortality enhancement in this infective system since experiments indicate that pulmonary deposition of organisms is less in ozone-treated animals (15).

As might be expected, pulmonary edema contributes to mortality from infections when exposure to edemagenic doses are employed. Thus in experiments reported by Coffin et al. (91), employing a tolerance inducing regimen about 50 percent protection was noted when exposures were to 588 $\mu g/m^3$ (0.3 ppm) O_3 and above. However, when exposures to 196 $\mu g/m^3$ (0.1 ppm) were employed, no such inhibition by the influence of tolerance was noted. The investigators' interpretation is that while edema plays a role in enhancing the infectious process at the higher concentration, there is still an increment of the infectious process related to the depression of the specific antibacterial mechanisms by ozone exposure. Furthermore, at concentrations below those eliciting pulmonary edema, no inhibition by tolerance indicates that the reason for the enhancement of the infectious process lies in other processes, i.e., cellular defenses and the like.

The possible role of the cellular components of the lung in enhancing infection has been the subject of a number of reports since the pulmonary macrophage is often considered the first line of defense against inhaled viable particles by virtue of its mobility, phagocytic properties, and enzyme potential. Exposure to ozone is very detrimental to alveolar macrophages. It inhibits both the number of phagocytes in the lavageable pool and their ability to phagocytize bacteria of the lung (4,92). The macrophages recovered by lavage also have diminished enzyme action, both in vivo and in vitro (38,93). Thus, they presumably have reduced power to engulf and kill bacteria (94,95). These changes are transient and most parameters are back to pre-exposure levels by 24 hours after a single exposure; it is known that longer, continued, intermittent exposure leads to increased numbers of macrophages as viewed in fixed sections (18). The flattening of the dose response curve in acute studies may be due to the influx of new macrophages since recent reports suggest a non-pulmonary origin for these cells (96). It is difficult to assess the importance of the polymorphonuclear leukocyte in the lung airway. Ozone is chemotactic and elicits the outpouring of numerous neutrophilic leukocytes into the airway at the termination of a 3-hour period of exposure and peaks 6 hours after exposure. Presumably, most of these leukocytes were subject to little exposure to ozone and thus would be in a good position to eliminate the bacteria. Such is obviously not the case, since bacteria persist and grow to enormous numbers in the presence of these leukocytes. It would thus appear that the polymorphonuclear leukocyte exerts little bacteriostatic effect in the lung airway of the ozone-exposed animal.

The anatomic lesions from both ozone and nitrogen dioxide exposure may be conducive to the establishment of bacterial infection. These lesions have been discussed in Section B. However, it is germane to this section to point out that the lesions induced in the cilia-bearing cells and the denudation of the basement membrane by desquamation of Type I alveolar epithelial cells might logically lead to slowed physical clearance of bacterial cells deposited in the tubular airways and increased replication and vascular penetration by bacteria deposited in the terminal airways deep in the lung.

There is some evidence that the effect on the pulmonary macrophage might be mediated through the non-cellular milieu of the lung airway. One possibility is that the pollutant inactivates an oposonogenic factor since it is known to have protein degrading properties (7). However, the macrophage exists in an environment in which a liquid layer covers the epithelial cells lining the alveoli and invests the macrophages (97). It is thus reasonable to speculate that ozone or reaction products react first with this lining film which in turn influences the macrophage deleteriously. Experiments conducted by Gardner et al. (66) on lavage fluid collected from rabbits, indicate that ozone exposure prior to the lavage reduces the stability in vitro as compared to control lavage fluid when normal macrophages are added. A similar effect is noted when ozone is bubbled through the lavage fluid in vitro. Since no significant alteration of the surface tension of the lavage fluid was produced by ozone exposure in these experiments, it is postulated that the effect is on a non-lipid moity, possibly a protein (15,18). This activity found in cell-free pulmonary lavage fluid has been designated "protective factor" (66).

4. Discussion

The infectivity model demonstrates striking differences between exposure to oxidants and control atmospheres. A number of factors are probably responsible for this effect which appears associated with both a diminution of initial bacterial decline and a subsequent rapid multiplication of bacterial cells within the lung. The depression of macrophage numbers, inhibition of phagocytosis, and loss of activity of the lysosomal enzyme probably contribute to increased susceptibility from exposure to ozone. Edema certainly plays a role at edemagenic doses (probably from 588 $\mu g/m^3$ or 0.3 ppm in the mouse) but enhancement of mortality occurs at concentrations below any demonstrable edema (down to 157 $\mu g/m^3$ or 0.08 ppm). The effect on the so-called "protective factor" may play a role in contributing to a loss of stability of the macrophage. The anatomical lesions which are essentially similar for both ozone and nitrogen dioxide may be contributory in paving the way for initiation of the lesion and possibly permitting invasion of the blood stream. The establishment of the infection certainly reflects the reduced resistance of the lung after exposure to ozone and nitrogen dioxide. It is thus an exquisitely sensitive biological indicator. In the case of ozone, at least since the infection can be enhanced for several genera of bacteria in several species of laboratory animals at concentrations of the gas well within ambient ranges, it appears doubtful that man would be incapable of reacting similarly providing a sufficient concentration of the gas was presented at the susceptible pulmonary surfaces. It would seem that in the absence of epidemiological surveys relevant to such associations, it will be necessary to rely on comparative exposure and mathematical modeling to determine the possible influence of such factors in human beings.

G. NON-PULMONARY EFFECTS OF OZONE

A number of so-called "systemic" effects have been ascribed to ozone exposure. These include changes in elements in the circulating blood; such as sphering of erythrocytes (98), chromosome breakage in lymphocytes (99), and retardation of deoxygenation of hemoglobin (47); alterations in the cell membranes and nuclei of myocardial cells; decrease in brain serotonin (8); increase of the RNA/DNA ratio in the liver (5); transitory increase in liver weight and alkaline phosphatase (11); affectation of the contralateral lung when the lungs were exposed unilaterally (60); and premature aging (79).

Many of these effects can be explained on the basis of local action of ozone within the lung; true systemic action need not be postulated. For instance, free radical strike or peroxidation may occur as the circulating erythrocites or lymphocytes pass through the pulmonary capillaries. If such a local action was the sole site of action for the chromosome breaks, for example, it would affect only mature lymphocytes which are end points in cell division and thus would have limited mutagenic significance as compared to actual injury of cells capable of mitotic activity within the various nidi of lymphocytic production. This is an important point that needs further elucidation. As far as the changes in erythrocytes are concerned, the sphering could be explained on the basis of ozone attacking the polyunsaturated lipids of the red cell envelope and stroma. The slowed deoxygenation might result from abnormal oxidation of the hemoglobin. Both could occur while the erythrocytes were passing through the lung capillaries. Changes in the RNA/DNA ratio, alkaline phosphatase, liver size, and the like might reflect stress and blood volume shift into the splanchnic area. It is possible that the edemagenic effect on the contralateral lung as reported by Frank et al. (60) may be artifactural on the basis of two points. The authors achieved their unilateral exposure state by producing one-sided pulmonary collapse by means of pneumothorax. Thus, there is a possibility that 1) there was some minimal ventilation due to imperfect collapse and consequent minimal ozone exposure, or 2) that artifactual alteration of the lung was induced by collapse and reinflation. Furthermore, their finding is not reported by Alpert et al. (61) who created the same sort of model, not by pulmonary collapse of the lung, but by unilateral exposure by means of differential catheterization. Two of the changes mentioned above, however, cannot on any basis be attributed to purely local chemical action within the lung or the production of a shock state by pulmonary irritation. Some circulating toxic factor must have been responsible if the changes in the heart muscle cells reported by Brinkman et al. (47) are valid. Two mechanisms appear theoretically tenable, i.e., 1) that a chain reaction involving free radicals which, once started in the capillary bed of the lung might continue long enough vis-a-vis the blood polyunsaturated lipid to reach distant organs (42), or 2) that the formation of fatty acid ozonide occurred in the lung. The latter appears an attractive alternative, since ozonide, once formed in the lung, might then gain admission to the blood and circulate for some time reaching all parts of the body exerting a highly toxic effect at any susceptible distant site.

The aging alterations reported for ozone exposure are of great interest, and, with what is now known of the chemistry of ozone, injury is to be expected. The possibility of a free radical effect has already been discussed. However, it is well known that ozone is highly active as a tissue oxidant. It not only oxidizes unsaturated fatty acid, but has strong oxidant action against thiols. Glutathione is oxidized and a significant portion cannot be again reduced by NADPH-glutathione reductase (36). Tocopherol is protective against many oxidative processes, including oxidation of polyunsaturated lipid, although apparently not against reduction of thiols (35). Ozone exposure is destructive to tocopherol and animals made artifically tocopherol deficient are more susceptible to ozone exposure (42,43). Harmon (100) has shown that the life of at least two strains of mice can be significantly prolonged by means of high antioxidant supplement of the ration. Thus, it might be tenable to postulate that ozone contributes to aging on the basis of accelerated oxidation.

H. OZONE AS A POSSIBLE MUTAGEN

Reference has been made in this chapter to chromosome breaks in lymphocytes in hamsters attributed to exposure to ozone and to the case for possible nonpulmonary effects. Hence, interest has been generated in the possible potential of ozone attacking the genetic material of the body on a systemic level as a mutagen. It follows, then, that if the effect of ozone is mediated via a free radical and if this action can be perpetuated distally from the lung, a strong possibility would exist that ozone could in fact act as a mutagen. The apparent similarity of certain effects of ozone to effects of ionizing radiation has also been aluded to as evidence for its being potentially a mutagen. All these things are merely circumstantial, however, and not actual proof of ozone's mutagenicity.

In order to act as a mutagen, a given agent must damage genetic material so that the abnormality results in a succession of cells with new genetic potential causing either anatomic or functional aberration in newly-formed cells within an individual or transmission of a genetic defect to a succeeding generation.

In vitro studies with ozone have shown its ability to change the adsorption spectrum of nucleic acids (101) and to alter pyrimidine bases of nucleic acids from *Escherichia coli* (102). It also has been claimed that *E. coli* exposed in water through which ozone had been bubbled developed specific mutants (103).

In vitro ozone exposure of chick embryo (104) and cultured human cells has produced chromosome aberrations that were apparently dose-related (105). Chromosome aberrations were noted in roots of *Vicia faba* (106) and, as mentioned previously, in hamsters' circulating lymphocytes following *in vivo* exposure of these animals to 392 $\mu g/m^3$ (0.2 ppm) O_3 for 5 hours (101). In the latter experiment, the ozone was generated by an ultra-violet light source situated within the exposure chamber, but shielded so that the animals had no direct irradiation by ultra-violet light. Two other experiments, both performed through *in vivo* exposure of mice are of interest. Brinkman et al. (98) exposed inbred grey mice and C57 blacks to 196 and 392 $\mu g/m^3$ (0.1 and 0.2 ppm) O_3 for 7 hours a day 5 days a week for 3 weeks prior to parturition. They noted an approximate four-fold increase in neonate mortality among the offspring from the ozone-exposed mothers.

Hueter et al. (80) exposed both male and female white Swiss mice to auto exhaust prior to breeding and continued exposure of the females in a experiment designed to determine the relative importance of the exposure on either sex (exposed males being bred to unexposed females, exposed females bred to unexposed males, exposed males bred to exposed females, and unexposed males bred to unexposed females). The females were examined for cervical plugs to ascertain the efficiency of breeding and uterine scars counted to ascertain the number of implantations. The data indicate that a diminution of litter size attributable to fewer implanations occurred from matings in which the mates had been exposed to ozone.

In view of the foregoing findings, it might be germane to cite somewhat similar exposure studies made with an oxidant containing atmosphere (irradiated auto exhaust smog). Hueter et al. found that female mice exposed to this atmosphere before, during, and after gestation showed a marked decrease in number of litters, total number of pups born, frequency of litters per mother, and survival of infant mice. Lewis et al. (107), in a study aimed at elucidating the above findings, explored the subject when mice were exposed in a factorially designed experiment. They found that pretreatment of males with irradiated auto exhaust smog and switching of the females from one atmosphere to another appeared to reduce the number of births. This phenomenon is apparently not associated with absorption of fetuses in utero or by a diminution of copulation. The investigators also claim that heightened infant mortality was associated with exposure of the males before mating. They speculate that their findings may have been due to alteration of the genetic composition or perhaps other cellular components of the sperm.

I. TOLERANCE TO OZONE

Many irritant inhalants have the property of stimulating "tolerance," a state in which the toxicity of a subsequent dose is blocked to a greater or lesser extent by a prior exposure to a lower, non-lethal, dose. So-called cross-tolerance develops between different irritants when administered in a tolerance-developing sequence, i.e., ozone against phosgene, or vice versa.

This phenomenon has elicited a great deal of toxicologic interest and consequently has been studied rather extensively. Ozone is a particularly strong tolerance-inducing pollutant, either against itself or through cross-tolerance with other agents. With ozone, tolerance begins to be initiated at 588 $\mu g/m^3$ (0.3 ppm), reaching its maximum protective effect at 5880 to 7840 $\mu g/m^3$ (3 to 4 ppm) for the initiating dose (108). Tolerance begins to develop within 30 minutes, reaches its maximum in approximately 24 hours, and is known to persist for several months or longer.

Several mechanisms of action have been proposed for tolerance development: 1) Mobilization of a protective chemical by the sensitizing dose. In this category might be mentioned histamine antagonists, GSH, acid hydrolases, or the induction of a specific enzyme, 2) Physical thickening of the alveolar walls as the result of edema fluid, 3) development of cellular resistance. None of the above mechanisms appear to satisfactorily explain tolerance in all its manifestations. Fairchild (109) has observed that lung GSH levels were maintained in ozone tolerant animals, but not in non-tolerant animals. This situation could be brought about either through the enzymic pathway which would stimulate augmented production of GSH in response to a second exposure to ozone or by directly blocking the oxidation of GSH by some means. It would appear that the cycle of depression and augmentation of GSH following a single exposure to ozone does not necessarily result in augmented GSH levels at the point where maximum tolerance should occur, i.e., 24 hours after exposure (36).

Another factor which might mitigate against the GSH protective mechanism or of other specific augmentation or induction of specific blocking agents on a molecular level is the extremely divergent chemical character of the various agents capable of eliciting cross-tolerance.

Also, in regard to GSH, it is entirely possible that the maintenance of these thiols in the tolerant animals was merely secondary to their protected state, rather than the cause of it. While the development of a histmaine release-blocking agent might be postulated as a consequence of pretreatment, the reappearance of histamine in the ozone exposed lung does not parallel the duration of tolerance.

Henshler (110) proposes that the physical swelling of the interalveolar septa due to pulmonary edema blocks the irritant effect. This is an attractive possibility, though it is difficult to justify by virtue of the persistence of tolerance for 14 weeks or longer as previously mentioned (108). Recent information based on studies of tolerance induction through unilateral lung exposure suggest that there is little or no protection unless the particular lung has actually been previously exposed to ozone, that is, no significant crossover occurs from the contralateral lung (60,61,111). This suggests that there is no basis for a circulating humoral agent and that the phenomenon may be a purely locally inspired mechanism.

The significance of tolerance in the protection of the population at risk against community air pollution may be questionable. In small rodents, there is a sharp reduction in the elicitation of tolerance when the initiating dose is below 3920 to 5880 $\mu g/m^3$ (2 to 3 ppm) and it appears to be extinguished at approximately 588 $\mu g/m^3$ (0.3 ppm) O_3 (108). This is approximately the cut-off point for production of demonstrable edema in rats by means of recovery of labelled albumin via lung lavage where the no effect level is between 490 and 980 $\mu g/m^3$ (0.25 and 0.5 ppm) O_3 for 6 hours (13). Thus, it may be necessary to administer a minimally edemagenic dose before tolerance to subsequent edema formation develops. The ozone injury that is blocked by tolerance may, therefore, be one that specifically induces development of pulmonary edema. Such injury is unlikely to occur in human beings casually exposed to ambient air. Experiments conducted on mice by Coffin et al. (91), in which the parameter of resistance to bacterial infection was the indicator of toxicity from ozone exposure, suggest that there is a biphasic response with partial protection above 588 $\mu g/m^3$ (0.3 ppm) O_3 and no protection at all below this concentration (91). Since it is known that the presence of edema fluid in the lung increases the risk of pulmonary infection, it seems reasonable to postulate that the portion of the reduction of mortality attributable to tolerance was a blocking of the edemagenic response. Furthermore, the non-affected increment in mortality (above 588 $\mu g/m^3$ or 0.3 ppm) and the lack of any tolerance below this point might be interpreted as the result of ozone action on cellular and non-cellular specific defense systems in which tolerance could not be induced. Experiments by Gardner et al. (111) indicate that while reduction in edema development could be induced by single or repetitive ozone exposures, no such protection was obtained for the chemotactic expression of ozone in the lung of the rabbit. This finding might be interpreted as suggestive of a dual mechanism, one blocked by tolerance (edema formation), and one not affected (influx of inflammatory cells), thus separating edema and inflammation on the basis of tolerance.

In the latter study, it was also apparent that there was no influence of tolerance on the ozone injury to the macrophage system as measured by numbers of recoverable cells in pulmonary lavage, in the phagocytosis index, or in alterations of the acid hydrolases.

It is probably germane to discuss another phenomenon which may or may not be related to tolerance, but which might be better termed an adaptive state. This was first noted by Freeman et al. (18) related to NO_2 exposure, but preliminary observations by the same group of investigators show that ozone exposure results in the same change. The sequences are similar when repetitive or continuous exposures are performed for either gas, although the ozone threshold is 10 to 20 times lower than that for NO_2 (28). Animals undergoing either continuous or intermittent exposure to 490 to 1764 $\mu g/m^3$ (0.25 to 0.9 ppm) O_3 show necrotic and desquamative changes of certain cells of the terminal portion of the bronchioles and proximally located alveoli within a few hours, and by 24 hours these cells are replaced by new cells which can be distinguished morphologically from their predecessors. In the pulmonary alveolus, the squamous or Type I epithelial lining cell is cast off and replaced by the granular pneumocyte Type II cell. Characteristic morphological differences also occur in the terminal bronchiolar epithelia. The sequence of the change has been characterized for NO_2 by means of studies performed by measurement of the rate of incorporation of radio-labelled thymidine in the newly-formed cells and observations of their eventual regression to the pre-exposed state (112,113). Two points of interest might be mentioned in this connection: 1) In the face of continuous or intermittent exposure to the same concentration of the gas, the desquamated cells are replaced by new types which eventually revert to a resemblance of the original; and 2) if the exposure is continued long enough, alteration in the stromal structure of the lung occurs with development of fibrosis and emphysema. Thus, in spite of the apparent adaptation of one cell type to the gaseous environment the change does not connote across-the-board protection of the lung. At this point, it is uncertain whether the adaptive response on the part of the lung cells is brought about by substitution of a more resistant normal cell for a less resistant one, or that some adaptation occurs on a molecular level. Pace (114) has described an adaptive response among cells in vitro in which resistance to a subsequent exposure to ozone was induced by prior exposure.

J. DISCUSSION OF EFFECTS OF OZONE

As can be seen, evidence is constantly accumulating that ozone is an extremely toxic gas. Data from early studies on laboratory animals indicated that death from pulmonary edema and inflammation occurred after exposure to concentrations of 29,400 to 58,800 μg/m^3 (15 to 30 ppm) O_3 and above for several hours and that pulmonary edema as measured by wet weight - dry weight ratios could be produced by exposure in the order of 1960 to 3920 μg/m^3 (1 to 2 ppm) O_3 for 3 hours in small rodents. Data reviewed here indicate that evidence of transudation of blood protein into the lung (as measured by recovery of labelled albumin in pulmonary lavage fluid) occurs after exposure of rats to between 588 and 784 μg/m^3 (0.3 to 0.4 ppm).O_3. Pathologic studies have demonstrated that ozone attacks all levels of the bronchial tree with microscopic lesions already evident after exposures to as little as 490 μg/m^3 (0.25 ppm) for a few hours. Cells are quickly damaged and cast off. Regeneration of the same or different cell types is apparent as early as 24 hours after exposure. This is probably not a specific lesion, but is indicative of the presence of a noxious influence at this anatomical level. There is a turnover of cells in the terminal portion of the bronchioles and proximal alveoli which produces a characteristic lesion consisting of enlarged cuboidal cells (hyperplastic Type II pulmonary epithelium). As mentioned previously, the tentative mathematical model for ozone uptake in the respiratory tract of man predicts that the peak dose would occur in the small, but non-terminal, bronchioles (16 to 20th segment of Weibel) whose constriction might be expected to lead to decrements in closing capacity measurements. It is of interest in this connection that concomitant exposure to sulfur dioxide and ozone in man causes exaggerated decrements in various pulmonary flow rate responses. However, such response might be predicted from a two-stage action, i.e, proximal airway reflex response for SO_2 and probably O_3 plus a second more distal airway direct tissue response for O_3. There appears to be some differences of opinion regarding the segmental level of the most severe airway response on the basis of pathologic anatomy in investigations conducted in laboratory animals: some investigators claim that the most sensitive tissue is in the terminal bronchioles and proximal alveolar epithelium, whereas, others think the higher airway is more sensitive. It is now known, however, that there is fairly diffuse damage throughout the pulmonary portion of the airway and the mathematical model mentioned above might not necessarily be completely accurate at this tentative stage since allowance for velocity of the bolus of gas at various segmented levels and other factors may need to be adjusted according to more precise pulmonary airway morphometry and experimental observations yet to be made. As previously mentioned, fibrosis and emphysema are features of exposure to ozone of longer duration either continuously or intermittently. The role of the epithelial lesion in the genesis of structural modification of the lung is unclear at this time. It may only, for instance, reflect a sequential cycle of epithelial injury and replacement, a common feature of more epithelial tissue to relatively minor injuries, or, on the other hand, it may be intertwined in some manner with the fibrotic and other changes important in the genesis of emphysema. It is tempting to speculate that the emphysema might be secondary to two independent factors which might act in concert, namely 1) partial obstruction of the distal airway by the maturation and contraction of the fibroplastic lesions in this region, and 2) loss of tone and stretching of the pre-existing collagenous pulmonary framework due to chemical alteration of the collagen molecule. Microscopic observations support the fibrosis of the distal airway. A report of the effect of O_3 (and NO_2) in the denaturation of collagen molecules has been referred to in Section D. Increased rigidity occurred principally at edemagenic exposures of ozone and presumably is associated with increased tissue pressure due to fluid or by dilution of the surface active material by edema fluid. Diminished recoil or elasticity demonstrated by pressure volume curves employing both air and saline on the other hand as noted with repeated exposures to low levels of ozone for 30 days suggest diminished contractile force of the lung. Furthermore, these same animals showed gross pulmonary overdistention and a noticeable, but according to the investigators, non-statistically significant reduction in number of alveoli.

It is possible that such changes herald the onset of pulmonary emphysema which develops at a later stage than elevated expiratory flow resistance. Furthermore, a non-reflex component may be involved. It is possible that the suspected non-reflex component is constriction of the lower airway in response to the direct ozone injury at this segmental level. The recent studies on human volunteers in which closing capacities were studied suggests that this level in the bronchiolar tree contributes significantly to physiologic alterations in human beings and that ozone acts reflexively by stimulating centers in the nasopharynx thus causing constriction of the larger airways and non reflexively by direct chemical action in the terminal portions of the airways. An interesting feature of acute health response of human beings to ozone exposure has been the apparent augmentation of effects when persons at risk in oxidant episodes indulged in sports. This has shown up in post hoc analysis studies of performance records of high school athletes (115), and in systematically conducted clinical observations of school children in Japan (116). A common feature has been that subjects exercising actively suffer higher risk. The observations that human volunteers suffer more decrement in pulmonary physiologic parameters and that mortality due to pulmonary edema (111) and susceptibility to pulmonary infection (88) when exercised during exposure to ozone tend to lend credence to these reports. It would appear fairly reasonable to suppose that the exercise factor produces an increased dose by virtue of increased minute volume. Another factor, however, may be peculiarly important in human exposure, that being mouth breathing during strenuous exercise since, as indicated under pulmonary physiology, uptake experiments conducted in anesthetized dogs suggest that a change from nose to mouth breathing would double the ozone dose delivered to the deeper airways. While a change from nose to mouth breathing is undoubtedly important, in man, this mechanism is not normally resorted to in exercised rodents in whom mouth-breathing is abnormal. Nonetheless, Stokinger (1) reports a several-fold diminution in the concentration of ozone required to kill rats exposed by exercising. Gardner et al (88) note an increment in mortality from bacterial infection after ozone exposure of mice while continuously walking.

There appears to be no question that exposure of mice to ozone and synthetic smog, at concentrations well below those seen in polluted ambient air enhances mortality from infections.

There is, however, a natural reluctance to relate such findings directly to human beings without substantiating evidence in a variety of species. Thus far, in limited studies, considerably higher concentrations of pollutants have been required to effectively show mortality differences in other animals. There is evidence that this may be related to the natural resistance of the animal species to the particular infectious agent rather than to a basic difference in its reactivity to the system. The important point for the successful operation of this system as presently used in animals is the availability of a microorganism that is capable of invading the lung with the production of subsequent mortality. The pollutant then enhances the mortality by depressing the bacterial defense mechanism.

In the application of this model to the human subject through epidemiological studies, it would appear essential to seek associations of the establishment of deep lung infection by such pathogens as *Diplococci pneumonia* or *Klebsiells pneumonia* with oxidant episodes. It would not appear that such studies have been seriously undertaken.

K. PEROXYACETYL NITRATES

1. Animal Data

Data on the lethality of peroxyacetyl nitrate (PAN) are sparse, but that which is available suggests that it is less lethal to mice than ozone, about the same as nitrogen dioxide, and more lethal than sulfur dioxide.

Campbell et al. (117) exposed mice to high concentrations of PAN, 480 to 700 $\mu g/m^3$ (97 to 145 ppm) as measured at the chamber outlet, for 2 hours at 27°C. The studies demonstrated that the majority of mice exposed to 540 $\mu g/m^3$ or 110 ppm or more, PAN died within a month. It was observed that mice exposed to higher concentrations died earlier than those exposed to lower concentrations. Median lethal exposures characteristically produced a delayed mortality pattern, with most deaths occurring in the second and third week after exposure. Mortality was greater among older mice than younger mice, and it was greater at higher temperatures. It was not influenced appreciably by changes in relative humidity.

2. Human Data

Experiments carried out on humans have suggested that exposure to PAN results in increased oxygen uptake during exercise. Smith (118) has carried out a group of studies on male college students averaging 21 years of age. The subjects were exposed to 1485 $\mu g/m^3$ (0.3 ppm) PAN by breathing through the mouth (nose clamps were used) for 5 minutes while at rest, and then the subjects were engaged immediately in 5 minutes of exercise on a bicycle ergotometer. Both air containing PAN and air free of PAN were used without the knowledge of the subjects. Since the pollutant has no characteristic smell or taste, it was considered that the experiment was carried out in a "blind" fashion. It was noted that there was a statistically significant increase in oxygen uptake during exercise, without any change at rest. Expiration velocity was reduced after exercise. The changes could possibly be a reflection of an increase in the work of breathing or due to an increase in airway resistance. Because the report of this work does not adequately describe the experimental design or the statistical analysis, these results merit replication before conclusive statements can be made.

3. Discussion

The data obtained so far on the effects of PAN in animals and man are too incomplete to enable conclusions to be drawn regarding any effects of this pollutant at ambient concentrations.

L. HYDROCARBONS

Experimental data (119) resulting from the exposure of animals and humans to various hydrocarbon compounds indicate that:

1. The aliphatic and alicyclic hydrocarbons are generally biochemically inert, though not biologically inert, and are only reactive at concentrations of 10^2 to 10^3 higher than those levels found in the ambient atmosphere. No effects have been reported at levels below 500 ppm.

2. The aromatic hydrocarbons are biochemically and biologically active. The vapors are more irritating to the mucous membranes than equivalent concentrations of the aliphatic or alicyclic groups. Systemic injury can result from the inhalation of vapors of the aromatic compounds; no effects, however, have been reported at levels below 25 ppm.

Aldehydes are one of the primary contributors to the eye irritation noted during photochemical smog; and, because there is a direct relationship between aldehydes and hydrocarbons as determined through various atmospheric reactions, a review of aldehyde toxicology is included. Experimental data indicate:

1. Aldehyde concentrations have been correlated with the intensity of eye irritation during natural and laboratory-produced photochemical smogs. Formaldehyde appears to be detectable by odor or physiological response (optical chronaxy) at concentrations in the order of 70 μg/m^3 (0.06 ppm). The threshold for eye irritation by formaldehyde has been estimated by various investigators to be between 12 and 1230 μg/m^3 (0.01 to 1.0 ppm). Acrolein can be detected by odor and eye irritation at concentrations as low as 600 μg/m^3 (0.25 ppm), but the threshold for optical chronaxy is reported at 1750 μg/m^3 (0.75 ppm).

2. Formaldehyde at a concentration of 2460 μg/m^3 (2 ppm) has been associated with a doubling of the total expiratory resistance of guinea pigs exposed to irradiated atmospheres of automobile exhaust gases.

3. Acetaldehyde is much less irritating than formaldehyde; its irritant threshold appears at concentrations of about 10,000 μg/m^3 (50 ppm). No physiological effects are known at levels that may be anticipated to occur in the ambient atmosphere.

In general, the most characteristic and important effect of aldehydes for both humans and animals is primary irritation of the eyes, upper respiratory tract, and skin. The unsaturated aldehydes are several times more toxic than the corresponding aliphatic aldehydes; toxicity generally decreases with increasing molecular weight within the unsaturated and aliphatic aldehyde series. Animal experiments have shown that aldehydes can affect respiratory functions, causing such effects as an increase in flow resistance and tidal volume and a decrease in the respiratory rate. Animal experiments have also indicated that acrolein and formaldehyde, in the presence of certain inert aerosols, appeared to be more toxic to mice than the pure compounds themselves.

Pulmonary function and eye irritation have also been evaluated by the exposure of experimental animals and humans to various mixed atmospheres that had the characteristic of providing the milieu necessary for hydrocarbon reactivity. Experimental data indicate:

1. Exposure of guinea pigs to ambient Los Angeles air may produce a temporary increase in pulmonary airflow resistance. Exposure of guinea pigs to laboratory-irradiated auto exhaust causes as increased tidal volume, minute volume, and flow resistance, with a decrease in the respiratory rate. These parameters return to normal immediately following exposure.

2. The precursors of the eye irritants are hydrocarbons (as well as other organic compounds) in combination with oxides of nitrogen; the alkylbenzenes and olefins are more effective precursors than paraffins, benzene, and acetylenes. The products of photochemical reactions that have been identified as effective eye irritants are formaldehyde, acrolein, peroxyacyl nitrates, and peroxybenzoyl nitrate.

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CHAPTER 7

EPIDEMIOLOGICAL APPRAISAL OF PHOTOCHEMICAL OXIDANTS

A. INTRODUCTION

The possibility that photochemical air pollution could be a major health hazard has been of growing concern. A number of systematic studies have been conducted in an attempt to determine if an association exists between episodes of high oxidant pollution and general mortality, acute illness, aggravation of chronic respiratory disease, impairment of performance, or untoward symptoms such as eye irritation. The purpose of this chapter is to review the data and conclusions of these studies. In some cases, the data have been reanalyzed. Areas of insufficient knowledge and the relevance of the existing data to air quality criteria are emphasized.

B. ACUTE EFFECTS OF PHOTOCHEMICAL OXIDANTS

To identify the acute health effects of photochemical oxidant pollution, observations of the same populations or communities are made during periods of both high-level and low-level pollution. These health effects are compared with short-term pollutant concentrations measured as 24-hour averages, hourly maxima, or instantaneous peak concentrations.

1. Daily Mortality in Relation to Variations in Oxidant Levels

a. Mortality Among Residents Age 65 Years and Older

A study of the relationship between daily concentrations of photochemical oxidants and daily mortality among residents of Los Angeles

County age 65 years and older was reported in March 1955, March 1956, and February 1957 (1). The number of deaths per day was related to 2 indices: daily temperature at the downtown weather bureau, and oxidant concentrations from August through November of 1954 and July through November of 1955 (Table 7-1 and Figure 7-1). Daily mortality clearly exceeded the average during a heat wave occurring in late August and early September of 1955. High concentrations of photochemical oxidants ocurred immediately before, during, and after the heat wave. Daily mortality decreased, however, when the temperatures dropped, whereas elevated oxidant levels persisted.

Thus the oxidant level did not independently affect daily mortality counts. An inconsistent pattern was observed during other months when daily mortality was compared with both temperature and oxidant gradients (Table 7-1). For example, on days when the maximum temperature fell to between 21 to 26°C, variations in oxidant levels from low to medium to high showed no consistent relationship to the average number of deaths. For the months covered by this study, therefore, daily mortality among persons age 65 years and older in Los Angeles County was strongly influenced by a heat wave, but was not altered consistently by variations in oxidant concentrations.

b. Mortality and Heat Waves

Since there is a meteorological association between high temperatures and high oxidant concentrations in the Los Angeles basin, the conclusions of the previous paragraph do not preclude a relationship between increased mortality and the simultaneous occurrence of high temperatures and high oxidant concentrations. That is, high oxidant

Table 7-1. AVERAGE NUMBER OF DEATHS PER DAY DUE TO CARDIAC AND RESPIRATORY CAUSES AMONG RESIDENTS OF LOS ANGELES COUNTY, AGE 65 AND OLDER, AS RELATED TO TEMPERATURE AND OXIDANT CONCENTRATIONS BY MONTH, 1954-1955 (1)

	Totals		Temperature readings											
			10°-15°C (50°-59°F)		16°-20°C (60°-69°F)		21°-26°C (70°-79°F)		27°-31°C (80°-89°F)		32°-37°C (90°-99°F)		>38°C(>100°F)	
Concentration, ppm	Number of days	Average number of deaths	Number of days	Average number of deaths	Number of days	Average number of deaths	Number of days	Average number of deaths	Number of days	Average number of deaths	Number of days	Average number of deaths	Number of days	Average number of deaths
August, 1954														
Low (00 - .24)	8	29.9					4	28.2	4	31.5				
Medium (.25 - .49)	22	32.2					7	30.1	12	31.8	3	39.0		
High (.50 +)	1	32.0					1	32.0						
September, 1954														
Low (00 - .24)	1	26.0					1	26.0						
Medium (.25 - .49)	25	32.1					5	29.6	16	35.1	4	36.0		
High (.50 +)	4	33.3							4	33.3				
October, 1954														
Low (00 - .24)	9	39.4			1	31.0	6	38.2	2	47.5				
Medium (.25 - .49)	14	37.1			1	43.0	10	36.0	2	39.5	1	37.0		
High (.50 +)	8	36.3					3	36.7	5	36.0				
November, 1954														
Low (00 - .24)	16	38.6	1	39.0	5	36.0	4	42.0	6	38.6				
Medium (.25 - .49)	14	37.9			4	36.8	5	34.8	5	41.8				
High (.50 +)														
July, 1955														
Low (00 - .24)	4	35.0					1	28.0	3	37.3				
Medium (.25 - .49)	11	34.8					5	34.4	6	35.2				
High (.50 +)	2	42.0					1	42.0	1	42.0				
No Readings	14	37.5					10	38.7	4	34.5				
August, 1955														
Low (00 - .24)	5	36.8							4	35.5			1	42.0
Medium (.25 - .49)	16	38.4							16	38.4				
High (.50 +)	4	37.2							4	37.2				
No Readings	6	37.8							4	38.5	2	36.5		
September, 1955														
Low (00 - .24)	5	29.0			1	33.0	3	27.3	1	30.0				
Medium (.25 - .49)	13	61.5					4	30.8	3	43.7	1	52.0	5	98.8
High (.50 +)	7	50.3					1	32.0	1	28.0	3	40.7	2	85.0
No Readings	5	34.8					4	33.2			1	41.0		

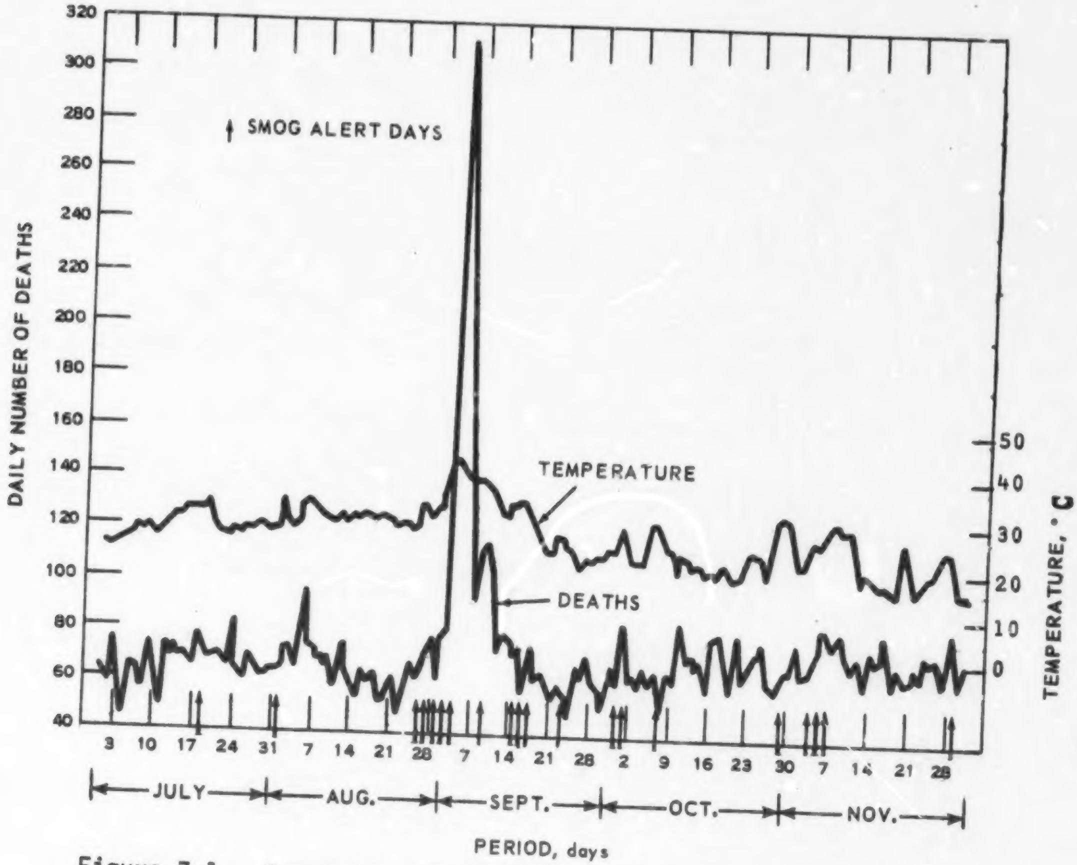


Figure 7-1. Comparison of deaths of persons, 65 years of age and over, and maximum daily temperatures, Los Angeles County, July 1 to November 30, 1955 (1).

concentrations may augment the mortality effect of high temperature. The results of a study by Oechsli and Buechley (2) of mortality associated with three Los Angeles heat waves--in 1939, 1955, and 1963--provide information on this multiple relationship. Daily mortality during these heat waves was compared with daily mortality occurring immediately before and after each heat wave and with mortality during the same season in 1947 when no heat wave occurred.

Significant increases in the mortality ratio were observed with increases in temperature during each of the three heat waves. Above-average deviations were greatest among the more elderly persons. Of the three incidents, the one occurring in 1963 is notable for a considerable diminution in excess mortality, even though temperatures reached the same peak levels as in 1939 and 1955. It has been surmised that the use of air conditioning was one of several possible reasons for the reduction in mortality in 1963. There was no discernible difference in the magnitude of the mortality effects of heat in 1939 and 1955. It can be assumed that considerably less photochemical oxidant pollution accompanied the 1939 heat wave than was present in 1955, although oxidant was not being measured in 1939. The comparison of the 1939 and 1955 heat waves suggests, under the above assumption, that high photochemical oxidant concentrations do not augment the mortality effect of high temperatures.

c. Mortality of Nursing Home Residents

An attempt was made to establish whether nursing home patients, many of whom were chronically ill and thought to be unusually susceptible to atmospheric pollution, experienced greater mortality on or immediately after days of high oxidant concentrations (1). Deaths and transfers to hospitals among residents of 16 Los Angeles nursing homes having a total of 358 beds were recorded for 1954. An unusually large number of patients appeared to have been transferred to hospitals following a particularly heavy episode of smog during one week of the study period. A larger study of the nursing home population was conducted from July through December of 1955, during which all such homes in Los Angeles County containing 25 or more beds were surveyed. The number of institutions ranged from 90 to 92, with a total of 3734 to 3826 beds. Daily mortality, the corresponding maximum daily temperature, and the occurrence of smog-alert days with ozone concentrations of 590 $\mu g/m^3$ (0.30 ppm) or higher are shown in Figure 7-2. The heat wave in late August and early September again showed a striking effect on mortality; at all other times, variations in daily mortality did not appear to be related to the occurrence of smog-alert days.

d. Two-Community Study

Massey et al. (3) compared daily mortality in two areas of Los Angeles County. These areas were selected for both similarities in

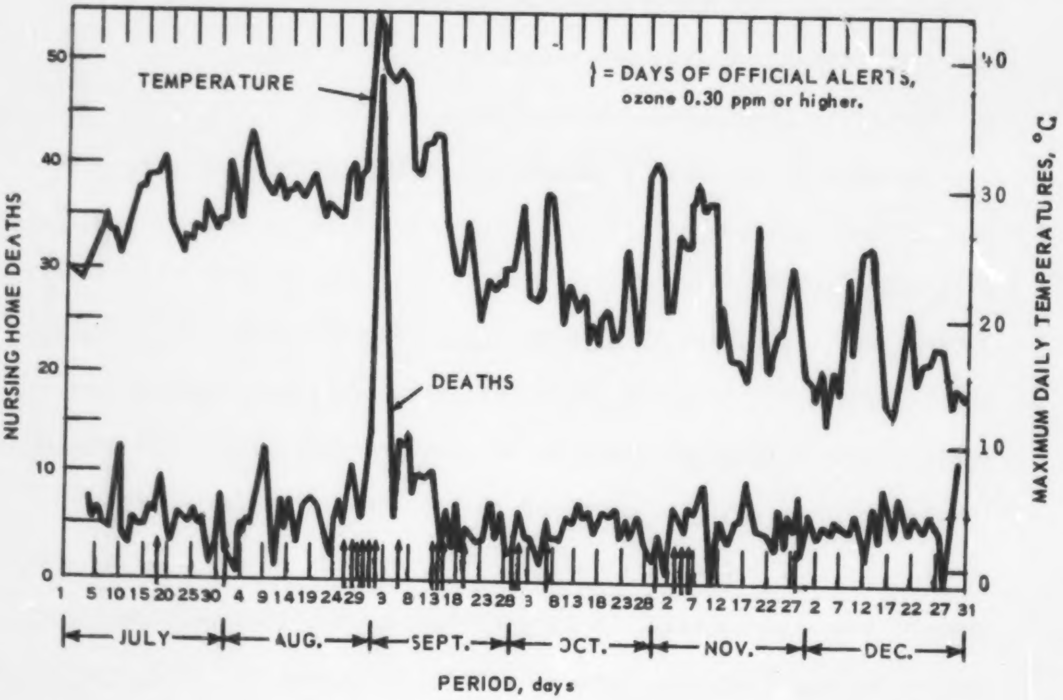


Figure 7-2. Comparison of nursing home deaths, maximum daily temperature, and "smog alert" days in Los Angeles County, July through December 1955 (1).

temperature and differences in air pollution levels. Two synthetic communities containing a combined population of 944,391 persons were thus formed. The pollutant variables used in this analysis were the daily maximum and mean oxidant levels, as established using the potassium iodide (KI) method, and sulfur dioxide and carbon monoxide concentrations. The synthetic communities were subdivided into smaller units, each of which was represented by a single temperature or air pollution station. The mean number of daily deaths in the low-pollution area was subtracted from the mean number of deaths in the high-pollution area, and the differences were examined by correlation and regression analyses with respect to differences in pollution. No significant correlations between mortality differences and differences in pollutant levels were observed.

e. Mortality from Cardiac and Respiratory Diseases

Hechter and Goldsmith (4) analyzed the effect of pollutant concentrations on average daily mortality from cardiac and respiratory diseases in Los Angeles County for the years 1956 through 1958. Daily mortality, averaged within each month of the study, fluctuated between 1.0 and 1.3 per 100,000 population. These fluctuations were approximately 180 degrees out of phase with fluctuations in oxidant and temperature values, and approximately in phase with maximum carbon monoxide concentrations (Figure 7-3). To remove the major effect of season of year, the authors fitted Fourier curves to the data and found a single cycle of Fourier functions to fit oxidant and temperature fluctuations in carbon monoxide and cardiorespiratory mortality (Figure 7-4). The residual variations from these fitted

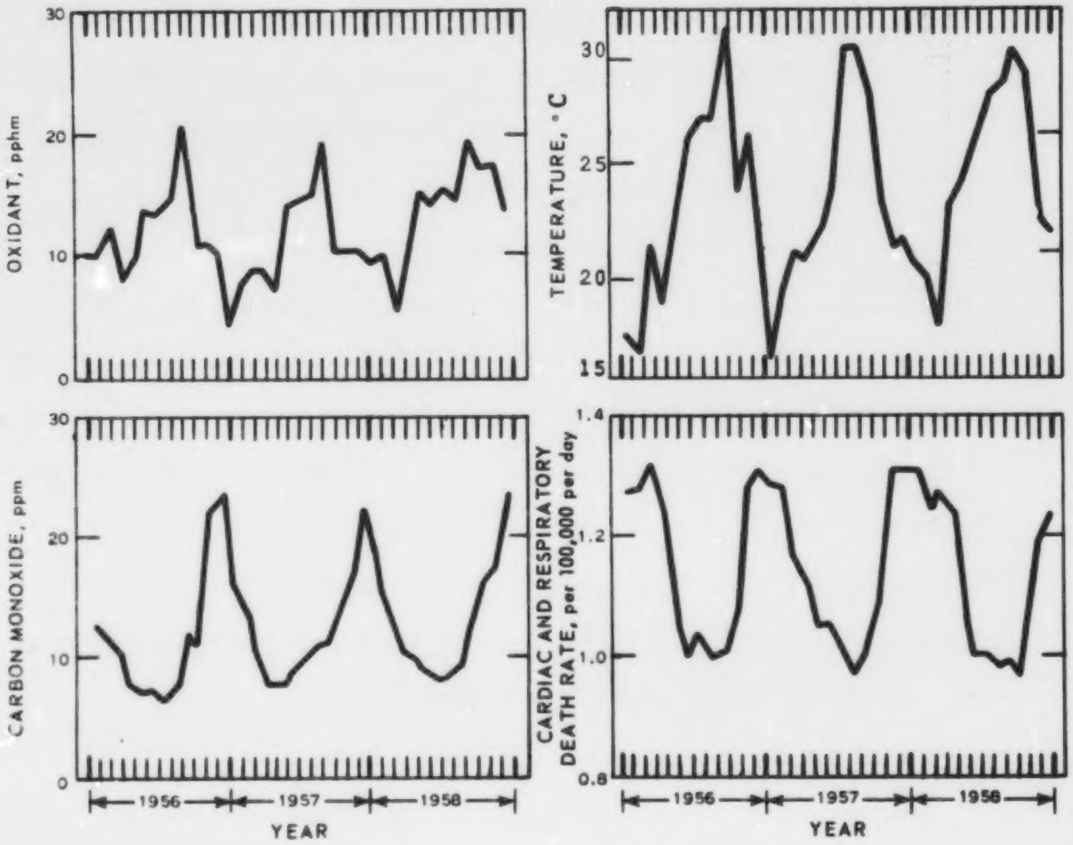


Figure 7-3. Comparison of maximum concentrations of oxidant and carbon monoxide, maximum temperature, and daily death rate for cardiac and respiratory causes, Los Angeles County, 1956-1958 (4).

curves for each of the variables were presumed to be independent of season. The relationship between pollution or temperature on one day with the value of the same variable on the successive or following days was also accounted for in the analysis. When residuals from the fitted curves were thus analyzed, no significant correlations between pollutants and mortality for cardiorespiratory diseases were found. Neither were there significant correlations when a lag of 1 to 4 days was applied.

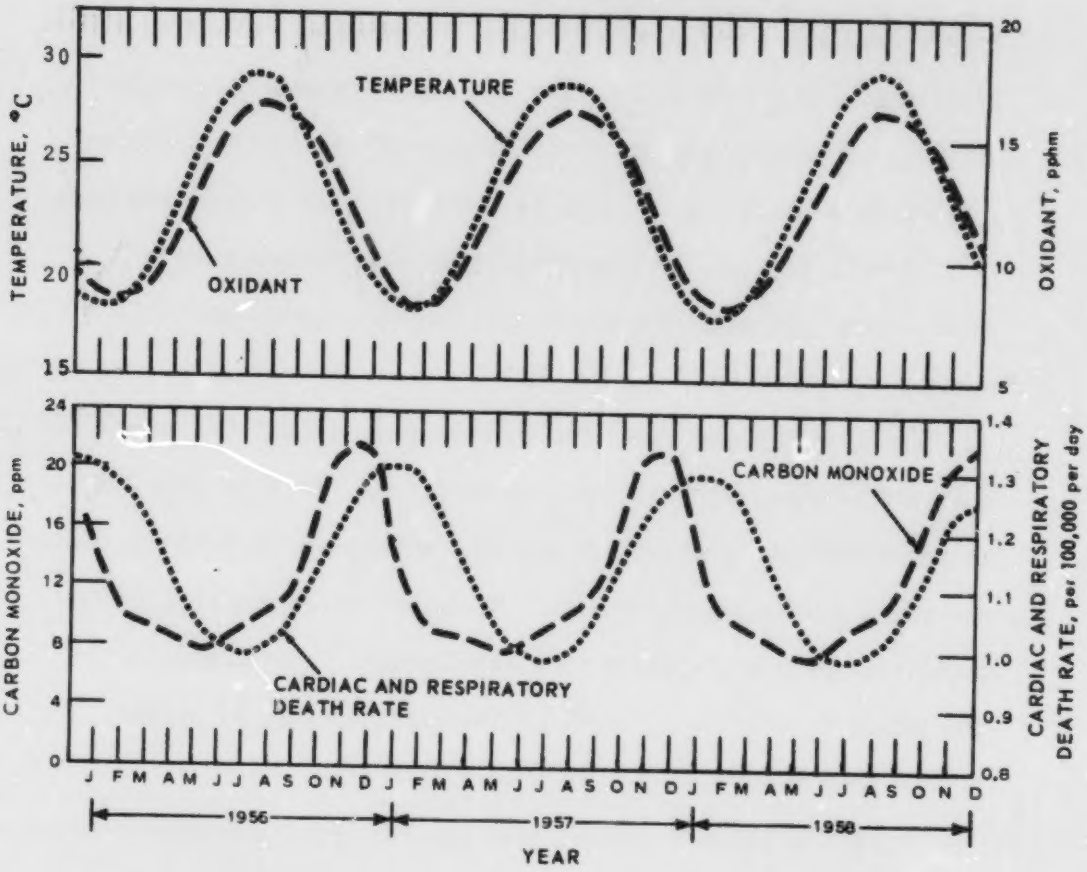


Figure 7-4. Fourier curves fitted to data in Figure 7-3 (4).

It should be noted that the analysis of mortality data by fitting them to Fourier curves derived from the same data may mask a real effect of environmental factors on mortality. In the absence of a simultaneous study of a less-polluted comparison community, no conclusive statement can be made regarding the effect of oxidant levels on community mortality.

Mills (5) compared cardiorespiratory deaths with the occurrence of smog in Los Angeles. A suggestive association between oxidant levels and excess deaths was observed on days when maximal oxidant levels rose above 390 $\mu g/m^3$ (0.2 ppm) measured as ozone (O_3). This relationship included deaths from influenza, pneumonia, bronchitis, asthma, and cardiac conditions. Analysis of seasonally corrected nursing home deaths in Los Angeles indicated that a close response relationship existed, with progressively more frequent deaths observed on days with maximal oxidant levels of 390 $\mu g/m^3$ (0.2 ppm) as O_3 and above.

While this paper attempted to take into account the impact of heat and seasonal variability upon mortality, there is some question as to whether the statistical analyses which were employed accounted for these confounding variables. Nevertheless, these results are suggestive of a mortality effect attributable to oxidants and indicate that additional study of this relationship is needed.

f. Discussion

A variety of methods have been used to examine whether short-term variations in photochemical oxidant concentrations have been associated with excess mortality in Los Angeles. The results have not consistently demonstrated such an effect. Studies comparing the mortality experience of Los Angeles with that of a less-polluted community are needed before firm conclusions can be drawn about the observed lack of pollutant-related mortality.

2. Hospital Admissions in Relation to Oxidant Levels

a. Los Angeles County Hospital Admissions, 1954

Admission records of Los Angeles County Hospital for the period September through December 1954 were examined in relation to several categories--namely, admissions for asthma in children under 10 years of age, for tuberculosis, for other respiratory conditions, and for other causes. Total admissions, the number of persons with acute conditions in all units, and the number who died were tabulated. No significant associations with oxidant levels were observed. Weekly admissions to an additional group of hospitals with a total of 2224 beds were similarly examined. The data appeared to show some seasonal trend, particularly for certain diseases of the cardiovascular or respiratory systems.

Brant (6,7) studied patients with respiratory or cardiovascular diagnoses who were admitted to or discharged from Los Angeles County Hospital between August 8 and December 25, 1954. Patients were excluded who were younger than 9 or older than 90 years of age, as were those who had not resided for at least 3 years in an area within 13 km of downtown Los Angeles. Meteorological data including temperature and relative humidity, were included in the analysis. Although the method of selecting patients for the study was not described, the selection process yielded 246 cardiovascular admissions and 122 respiratory admissions. Total oxidant, as measured about 1.6 km from the hospital, was used as an index of air pollution. Multiple regression analyses were used to relate hospital admissions to atmospheric and meteorological variables. Calculations included regressions of hospital admissions as late as 4 weeks after the occurrence of a given set of environmental measurements, and a significant correlation between periods of high oxidant, low relative humidity, and low temperature, and hospital admissions weeks later for cardiovascular conditions was observed. Because there is no biologically plausible explanation for this correlation, the association must be considered fortuitous. In contrast, there was a negative correlation between admissions for cardiovascular disease and oxidant levels either on the day of admission or for 2 weeks prior to admission.

b. Hospital Admissions in the Los Angeles Metropolitan Area

Based on hospitalization insurance records, Sterling et al. (8,9) assembled data from Los Angeles hospitals for the period of March 17 to October 26, 1961. Diagnoses were grouped according to "highly relevant" disorders. Classified as "highly relevant" were allergic disorders, inflammatory diseases of the heart, rheumatic fever, vascular diseases, and other diseases of the respiratory system. All other illnesses were classified as "irrelevant."

The authors found that the mean number of admissions varied by day of week. They were higher in the first half than in the second half of the week. They also observed that 1) oxidant precursors were exceptionally low on Sundays; 2) both oxidant and ozone levels were low on Saturdays and Sundays; and 3) nitric oxide was exceptionally low on Wednesdays. After correcting for the day of the week with respect to pollutant and admission frequencies, "highly relevant" illnesses were found to show significant correlations with oxidant levels, carbon monoxide, and ozone. "Highly relevant" conditions were also correlated with oxides of nitrogen and particulate matter. No significant correlations between hospital admissions and temperature were found, in contrast to the finding discussed in Section B.1 and in Figure 7-3, although only data for March and April were used. Using the same data, Sterling et al. (9) studied the association of duration of hospital stay and air pollution. The correlations were not statistically significant.

Because of both the limited period of the year utilized for study, and the extremely low correlation coefficients, it is difficult to conclude that a meaningful relationship has been demonstrated by these studies.

c. Discussion

Studies reviewed in this section have failed to demonstrate adequately any relationship between daily hospital admissions and variations in concentrations of photochemical oxidants.

3. Aggravation of Respiratory Diseases by Oxidant Pollution

a. Aggravation of Asthma

As a result of reports by physicians that asthmatic attacks are frequently associated with smog periods, Schoettlin and Landau (10) undertook a study to determine whether such a phenomenon does take place. Five physicians selected 157 patients, 137 of whom participated in the study. All resided and worked in the Pasadena area. Fifty-four of the patients were younger than 15 years of age. Most had had asthma for at least 5 or more years. Daily records of the time of onset and severity of asthmatic attacks were maintained by the patients for the period September 3 through December 9, 1956, since relatively high air pollution levels usually occur during these months of the year. The data were collected weekly. In the event a subject neglected to submit a report, the investigators telephoned and requested one.

The Schoettlin and Landau study as published is vague with respect to methods and time durations of pollution measurements. In the document, Air Quality Criteria for Photochemical Oxidants (AP-63), published by the U. S. Department of Health, Education, and Welfare (11), it was assumed that oxidant levels in this study were daily instantaneous peak values as measured by the phenolphthalein method. Errata to that document were issued indicating that oxidant levels in this study were, in fact, determined by the KI method and not the phenolphthalein method (12,13). There still remains confusion as to whether effects in this study were related to instantaneous peak values, which would be equivalent to maximum hourly average oxidant levels approximately one-half as great. A comparison of instantaneous peak oxidant readings with maximum hourly oxidant readings at monitoring station 4 of the Los Angeles County Air Pollution Control District in Pasadena during the period of this study (13) indicates that daily peak values of 490 $\mu g/m^3$ (0.25 ppm) calculated O_3 would have been equivalent to maximum hourly averages of 392 $\mu g/m^3$ (0.20 ppm) and 235 or 255 $\mu g/m^3$ (0.12 or 0.13 ppm) as had previously been concluded. In the discussion to follow, it is being assumed that oxidant levels reported in this study correspond to daily maximum hourly levels and not instantaneous peak levels, recognizing the small error that might result if readings had been instantaneous peaks instead.

The study showed that the peak period for asthma attacks was between midnight and 0600 hr, whereas the maximum oxidant levels were recorded between 1000 hr and 1600 hr. A delay in response to the inciting agent might explain this late period for asthma attacks. Of the 3435 attacks reported, fewer than 5 percent were spontaneously associated with smog by the patients, and none of these were severe. One-third of the attacks spontaneously associated with smog were reported by a single patient. The correlation coefficient between the number of attacks per day and the maximum oxidant reading was 0.37. Addition of other variables to the analysis did not significantly alter these results. There was no significant difference in the average number of attacks per day for days above the median maximum oxidant level 250 $\mu g/m^3$ (0.13 ppm) O_3 by the KI method compared with days below the median. There was, however, a significant increase in the mean number of attacks on days when daily maximum oxidant levels exceeded 490 $\mu g/m^3$ (0.25 ppm) by the KI method, contrasted with days when the daily maximum oxidant level fell below this level. The authors suggested that this may indicate a threshold level for oxidants above which there could be a physiologic response. There was also a significant association between attack rates on days in which plant damage occurred, in contrast to days without plant damage. Specific oxidant levels associated with plant damage were not reported in this study, however. This effect was most pronounced for persons who had lived in the area 10 years or more. The data were examined further to see whether a small number of the subjects might be responsible for the observed correlation. Eight individuals, or 6 percent of the total panel, were identified whose attacks corresponded most often to days on which plant damage occurred, but there was no attribute common to these individuals. Thus a small portion of people suffering from asthma appeared to be responsive to levels of photochemical oxidants sufficient to cause vegetation damage.

b. Aggravation of Emphysema and Chronic Bronchitis

Several studies have been conducted to determine if air pollution aggravates the condition of subjects suffering from chronic bronchitis and emphysema.

Motley et al. (14) reported on the results of lung-function tests on 66 volunteers, 46 of whom had pulmonary emphysema. Lung-function tests were performed on subjects in rooms from which the oxidants were removed by activated charcoal filters. Twenty-one subjects stayed in the filtered rooms for periods between 2 and 4 hours, 20 subjects between 18 and 20 hours, and 25 subjects between 40 and 90 hours. No accurate measurements of oxidants were obtained but, during the periods of the study, oxidant concentrations ranged from 390 to 1370 $\mu g/m^3$ (0.2 to 0.7 ppm) as O_3 and ozone from 390 to 1040 $\mu g/m^3$ (0.2 to 0.53 ppm) at monitoring stations several kilometers from the chamber. Air was classified as smoggy when there were a definite odor of ozone, reduced visibility, eye irritation, and the prediction of smog by the Los Angeles Air Pollution Control District. Lung-function measurements were made of vital capacity, $FEV_{3.0}$, and maximal breathing capacity. Residual volume and air distribution measurements were also recorded. An improvement in lung function was observed, particularly a decrease in the residual lung volume in emphysematous subjects who remained in the chamber for 40 or more hours and who entered it on smoggy days. No significant changes in lung-volume measurements were obtained when normal subjects breathed filtered air. No significant changes were observed when emphysematous subjects entered the chamber on non-smoggy days. Variations in the smoking habits of test subjects could have significantly influenced the results, but no information on such variations was reported.

At Los Angeles County Hospital, Remmers and Balchum (15) utilized a room with an air conditioning system and a filter which could be used at the discretion of the investigator to remove photochemical oxidants, ozone, and nitrogen oxides from ambient air and partially remove particulate matter. Studies were conducted in September and October of 1964 and from March to November of 1965. Subjects performed lung-function tests one or more times daily while they lived in the room. In general, they spent 1 week in the air in the room without air filtration, and 1 week in the air in the room with filtration. During both weeks, air conditioning was adjusted to maintain a room temperature of 22 ± 1.4°C and a relative humidity of 50 ± 5 percent. A third week was spent in a room arranged for exposure to ambient atmosphere. Respiratory function tests of airway resistance, diffusing capacity, other pulmonary function tests, blood oxygen tension, and oxygen consumption during exercise were performed. Several tests were repeated while the patients were exercising. Because of the elaborate nature of the experimental system, only a small number of subjects could be studied. Eleven subjects were cigarette smokers, and one stopped smoking when he entered the study. Most subjects had moderate to severe respiratory impairment from bronchitis or emphysema. Hence, any impairment of respiratory function could be interpreted as a fairly serious deterioration of pulmonary status.

Preliminary examination of the data indicated that airway resistance was affected by elevated oxidant concentrations when observations were made over a range of 100 to 450 $\mu g/m^3$ (0.05 to 0.23 ppm). Determination of a threshold level for this effect cannot be made from these data because of the infrequent observations over the entire range of exposure. The subjects were also exposed to ambient air containing not only oxidants, but other substances as well. It is possible that the observed effects were caused partially by removal of other pollutants such as aerosols, particulates, PAN, or aldehydes. Cigarette smoking also varied and apparently was more frequent when the ambient air was not being filtered, thus possibly confounding the results. Another test which seemed to show significant changes with air pollution was the consumption of oxygen during exercise. Alterations in rate and depth of breathing could also have accounted for some of these effects, but these possibilities were not investigated.

Further analyses of these data were carried out for purposes of this report, and the results are shown in Table 7-2.

In view of the possible influence of cigarette smoking on these test results, data for cigarette smokers must be interpreted cautiously. The majority of the nonsmokers in this study did show decreases in airway resistance corresponding to decreases in oxidant exposure, but observations were far too infrequent for inferences to be drawn about a possible threshold level for this effect.

Table 7-2. CORRELATION OF MORNING AND EARLY AFTERNOON OXIDANT LEVELS WITH OXYGEN CONSUMPTION AND AIRWAY RESISTANCE OF 15 PATIENTS WITH CHRONIC RESPIRATORY DISEASE (15).

Patient's number and smoking history[e]	Number of observations		Maximum breathing capacity, liters/min.	Observed correlation coefficients				
				Oxygen consumption[a]			Airway resistance[b]	
				Resting conditions		Exercise conditions		
	Oxygen consumption	Airway resistance		a.m. oxidants	p.m. oxidants	p.m. oxidants	a.m. oxidants	p.m. oxidants
102 S	11	17	–	.282	.405	.774	-.379	-.313
[103 NS	14	14	–	.123	.210	.251	.717	.567
104 NS	14	14	–	-.313	.007	.258	.638	.641
106 S	17	17	88.4	.473	.579	.521	-.361	.146
[107 S	17	17	88.4	.473	.579	.521	-.361	.146
108 S	18	18	99.4	.489	.448	.409	-.378	.656
110 S	14	16	69.1	.255	-.136	-.172	.431	.124
[111 NS	15	17	117.7	.434	.092	-.459	.251	.354
112 S	15	15	52.9	.413	.209	-.348	.339	.433
[113 NS	12	12	181.5	-.107	.222	.088	.034	.006
114 S	13	13	65.3	-.114	-.158	-.120	-.161	.058
[115 S	14	14	26.3	.423	.290	.130	.217	.557
116 S	14	15	96.1	.288	-.094	.751	.715	.460
[117 S	15	15	38.6	–	.189	.456	–	.609
118 NS	15	15	37.4	–	.345	.138	–	.453
	"z"[f]			2.991[g]	2.761[g]	2.837[g]	3.621[h]	4.976[h]

[a] The first two correlations are with "resting" oxygen consumption, which was measured around 11 a.m.; the third correlation is with "exercise" oxygen consumption, which was measured around 3 p.m.

[b] Airway resistance is "resting"; values given are averages of four measurements made throughout the day

[c] a.m. Oxidant was measured around 9:30 a.m.

[d] p.m. Oxidant was measured around 1:30 p.m.

[e] Smoking History: S = smoker, NS = nonsmoker, SS = stopped smoking when study began. [- Indicates patients were tested during the same period.

[f] "z" values were found by converting the individual correlations to t values, using the relationship

$t_i = r_i\,[(n_i-2)/(1-r_i^2)]^{1/2}$ then summing the t values over all patients; the sums have variance

$V(\Sigma\, t_i) = \Sigma\,[(n_i-2)/(n_i-4)]$. The "z" values shown are the ratio of

$\Sigma(t_i)\,/\,\sqrt{V\,\Sigma_i\, t_i}$ which is approximately N(0,1).

[g] Significant at the 0.01 level.

[h] Significant at the 0.001 level.

Rokaw and Massey (16) conducted a study of the effects of environmental variables on pulmonary function in a group of 25 patients in a chronic disease hospital in Los Angeles over a period of 18 months. All of the patients had chronic, nontuberculous respiratory diseases, predominantly pulmonary emphysema. Each subject underwent a series of pulmonary function tests four times weekly; in addition, functional residual capacity by the helium dilution method was determined monthly. Air pollution data were obtained from a station about 0.4 km upwind from the hospital. Statistical methods of analysis were employed to detect associations between changes in pulmonary function and air pollution levels. The results showed a marked variability in the performance of pulmonary function tests; the variability was greater in the group of patients than in normal subjects; no such response was observed during major smog episodes, and no seasonal pattern of performance for the group was observed. [This is in contrast to the findings of McKerrow (17), who found yearly cycles in ventilatory function tests in a group of ex-miners with pneumonconiosis working in a car-assembly plant in South Wales.] The general level of oxidant in the hospital area was not as high as in some parts of the Los Angeles Basin ; the mean oxidant was 120 $\mu g/m^3$ (0.06 ppm), with a maximum of 820 $\mu g/m^3$ (0.42 ppm). It is possible that the experiment did not reveal any effect of oxidant on simple respiratory function tests because: 1) the group was too sick to respond ; 2) fluctuations in the subjects' disease states or cigarette smoking habits were the major determinants of test performance; or 3) the subjects were exposed to a relatively low level of oxidant.

Shoettlin (18) studied the long-term effects of community air pollution, occupational exposure to air pollution, and smoking among armed forces veterans living in the Domiciliary Unit and Chronic Disease Annex of the Los Angeles Veterans Administration Center. Day-to-day variations in the physical status of men with chronic respiratory disease were studied in relation to changes in environmental conditions in the coastal area of the Los Angeles Basin. Two groups of men were selected. The first group, consisting of 528 veterans who had no positive signs or symptoms of respiratory disease, was used as a control. The second group of 326 men was selected on the basis of the presence of at least two symptoms of respiratory disease (including cough, sputum, shortness of breath, and wheezing) for 2 or more years, and the presence of abnormal breathing sounds. Men from these two groups were matched by age and smoking history, and the resulting pairs were studied weekly by means of repeated pulmonary function tests and responses to a respiratory symptom questionnaire. About 65 percent of the men selected cooperated in the study. An air pollution monitoring station was set up at the site, and a companion program of observing vegetation damage was established. Analysis of variance showed no statistically significant effects of air pollution on respiratory symptoms or functions. The maximal oxidant values and oxidant precursor values, however, consistently accounted for more of the variation in frequency of symptoms and clinical signs for the diseased group than for the control group (Table 7-3). For example, the maximal oxidant precursor accounted for 30 percent of the variation in symptoms for the

Table 7-3. PROPORTION OF VARIATION ASSOCIATED WITH ENVIRONMENTAL FACTORS IN SYMPTOMS, SIGNS AND VENTILATORY TESTS IN A DISEASED AND IN A CONTROL GROUP (18)

Environmental factors	Diseased group			Control group		
	Symptoms	Signs	Puff-meter	Symp-toms	Signs	Puffmeter
Mean oxidant	0.11	0.13	0.04	0.08	0.06	0.04
Maximal oxidant	0.17	0.16	0.10	0.04	0.03	0.06
Maximal oxidant precursor	0.30	0.17	0.17	0.04	0.07	0.09
Pollen count	0.02	0.05	0.05	0.07	0.02	0.10
Maximal temperature	0.05	0.03	0.12	0.09	0.06	0.19
Maximal relative humidity	0.05	0.05	0.10	0.23	0.08	0.06

diseased group and 17 percent of variations in the Puffmeter test, whereas the proportion of the variation accounted for in the control group was insignificant.

Peters (19) examined pulmonary function in shipyard workers (welders, pipecoverers, and pipefitters) exposed to a variety of pollutants including metal fumes, asbestos, and nitrogen oxides as well as ozone. Evidence was found that chronic exposure to oxides of nitrogen and ozone among welders favors development of obstructive lung disease, whereas chronic exposure to asbestos among pipecoverers is associated with development of restrictive lung disease. When pulmonary function for these shipyard groups was compared to ventilatory function in pipefitters engaged in new-ship construction with minimal or no exposure to asbestos or welding fumes, the new-shipyard groups were found to be healthier. These data suggest, but do not prove conclusively, that chronic exposure to ozone, generally within acceptable industrial limits, may contribute to the development of obstructive lung disease.

c. Symptom Reporting in Student Nurses

Hammer et al. (20) examined symptom reporting in student nurses in relation to photochemical oxidant exposure. Freshman nursing students at the Los Angeles County General Hospital and the California Lutheran Hospital in Los Angeles were invited to participate in a prospective study of viral respiratory disease. In an effort to minimize bias, neither faculty nor students were informed that effects of air pollution were of major interest. Air quality was measured at a monitoring station located 1.5 to 3.0 km from both hospitals. Students in this study were followed through graduation. Daily symptom diaries were collected weekly and included questions related to such symptoms as headache, eye discomfort, cough, and chest discomfort. Symptoms were graded mild, moderate, or severe. Symptom reporting covered the period October 1961 through June 1964.

Patterns of symptom reporting from both schools were similar and were pooled in all analyses. All three grades of symptoms were considered positive responses because the occurrence of moderate or severe symptoms was rare. Pollutant-specific threshold funtions were calculated for each symptom. The hypotheses tested included the following: 1) that symptom reporting for headache, eye discomfort, cough, and chest discomfort was positively related to photochemical oxidant pollutant levels; 2) that symptoms were related most strongly to photochemical oxidants; and 3) that thresholds could be calculated for each symptom in relation to photochemical oxidants.

Simultaneous daily measurements for oxidants, carbon monoxide, nitric oxide, nitrogen dioxide, sulfur dioxide, and maximum daily temperature were available on more than 90 percent of the 868 days in this study. Simple headache frequency did not vary with oxidant exposure. The frequency of eye discomfort, however, increased as daily maximum hourly photochemical oxidant levels exceeded 294 to 372 μg/m^3 (0.15 to 0.19 ppm) (Table 7-4). Cough and chest discomfort both remained relatively consistent until maximum hourly oxidants reached 588 to 764 μg/m^3 (0.30 to 0.39 ppm), after which rates for both increased markedly. Temperature-fever adjusted symptom rates, except for headache, showed similar results with increases in oxidant exposure (Table 7-5). Temperature-fever adjusted headache rates increased slightly at daily maximum hourly oxidant levels of 196 to 274 μg/m^3 (0.10 to 0.14 ppm) and increased markedly as oxidant levels reached 588 to 764 μg/m^3 (0.30 to 0.39 ppm).

The relative increase for each symptom as a function of oxidant exposure is shown in Table 7-5 for headache, eye discomfort, cough, and chest discomfort. Hockey-stick threshold functions were computed for all symptoms and are shown in Table 7-6. Except for headache, the threshold and confidence limits for each symptom pair (simple and temperature-fever adjusted) were similar. The threshold for increased cough and chest discomfort was somewhere between a daily maximum hourly oxidant level of 500 to 600 μg/m^3 (0.25 to 0.30 ppm), whereas the threshold for increased eye discomfort was about 300 μg/m^3 (0.15 ppm). Symptom frequencies in this study were more closely related to photochemical oxidants than either carbon monoxide, nitrogen dioxide, or daily maximum temperature. The observed threshold for eye discomfort was comparable to that observed from other published reports. The observed threshold for cough and chest discomfort

Table 7-4. RELATIONSHIP OF AVERAGE DAILY PERCENT OF SIMPLE AND ADJUSTED SYMPTOMS TO PHOTOCHEMICAL OXIDANT LEVELS (868 DAYS, NOVEMBER 1961 THROUGH MAY, 1964)

Daily maximum hourly oxidant level, ppm[a]	Number of Days	Average number of nurses reporting daily	Average daily percent of symptoms reported							
			Headache		Eye discomfort		Cough		Chest discomfort	
			Simple	Adjusted[b]	Simple	Adjusted[b]	Simple	Adjusted[b]	Simple	Adjusted[b]
0.04	229	64	16.5	10.5	8.6	5.0	12.7	9.1	3.5	1.8
0.05-0.08	184	59	16.3	10.7	9.2	5.4	13.4	9.9	3.6	1.8
0.09	35	58	16.0	10.6	9.0	5.6	13.3	10.2	3.2	1.9
0.10-0.14	176	62	15.6	11.0	8.6	5.9	11.9	9.4	3.4	1.8
0.15-0.19	144	58	15.7	11.4	10.0	6.9	12.3	9.7	3.7	1.7
0.20-0.24	63	60	15.6	11.6	12.2	9.2	11.6	9.1	3.1	1.6
0.25-0.29	25	60	16.7	11.5	14.9	11.2	12.4	9.6	3.6	2.0
0.30-0.39	9	67	16.9	13.4	21.1	17.8	15.2	11.7	4.1	2.3
0.40-0.50	3	53	16.8	15.0	35.0	31.8	19.3	16.9	7.0	5.8
Overall average		61	16.1	10.9	9.6	6.3	12.6	9.5	3.5	1.8

[a] 1 ppm = 1960 μg/m³

[b] All days on which the symptom was reported along with "feverish", "chilly", or "temperature" are excluded.

Table 7-5. RELATIVE INCREASE OF HEADACHE, EYE DISCOMFORT, COUGH, AND CHEST DISCOMFORT IN RELATION TO PHOTOCHEMICAL OXIDANT EXPOSURE

Daily maximum hourly oxidant level, ppm[a]	Relative increase of symptom							
	Headache		Eye discomfort		Cough		Chest discomfort	
	Simple	Adjusted[b]	Simple	Adjusted[b]	Simple	Adjusted[b]	Simple	Adjusted[b]
≤0.08	1.00(16.4)[c]	1.00(10.6)	1.00(8.9)	1.00(5.2)	1.00(13.0)	1.00(9.5)	1.00(3.5)	1.00(1.8)
0.09	0.97	1.00	1.01	1.07	1.02	1.07	0.91	1.06
0.10-0.14	0.95	1.03	0.96	1.13	0.91	0.99	0.97	1.00
0.15-0.19	0.95	1.07	1.12	1.33	0.95	1.02	1.05	0.94
0.20-0.24	0.95	1.03	1.37	1.77	0.89	0.96	0.89	0.89
0.25-0.29	1.01	1.07	1.67	2.15	0.95	1.01	1.03	1.11
0.30-0.39	1.03	1.26	2.37	3.42	1.16	1.23	1.17	1.27
0.40-0.50	1.02	1.41	3.93	6.11	1.48	1.77	2.00	3.22

[a] 1ppm = 1960 μg/m³.

[b] Excludes all days when the symptom was reported in conjunction with either "feverish", "chilly", or "temperature."

[c] Baseline symptom rate as average daily percent of symptom reported

Table 7-6. HOCKEY-STICK THRESHOLD FUNCTIONS FOR SIMPLE AND ADJUSTED[a] SYMPTOMS IN RELATION TO PHOTOCHEMICAL OXIDANTS

Symptom	Oxidant[b] threshold		95% confidence limits				Baseline daily	Slope of
			Lower		Upper			
	$\mu g/m^3$	ppm	$\mu g/m^3$	ppm	$\mu g/m^3$	ppm	Symptom rate, %	increase, %
Headache								
Simple	--		--		--		--	--
Adjusted[a]	100	0.05	None		390	0.20	10.5	0.00003571
Eye discomfort								
Simple	310	0.16	280	0.14	340	0.17	8.8	0.0003469
Adjusted[a]	300	0.15	260	0.13	330	0.17	5.4	0.0003316
Cough								
Simple	510	0.26	370	0.19	None		12.5	0.0001632
Adjusted[a]	530	0.27	400	0.20	None		9.5	0.0001785
Chest discomfort								
Simple	580	0.30	350	0.13	None		3.4	0.0000918
Adjusted	530	0.30	420	0.21	None		1.7	0.0001020

[a] All days on which the symptom was reported along with "feverish", "chilly", or "temperature" are excluded.

[b] Thresholds are computed as maximum daily 1-hour levels of photochemical oxidants.

is consistent with studies showing effects of ozone upon pulmonary function and increased irritation symptoms among healthy males performing intermittent light exercise at exposures of 725 $\mu g/m^3$ (0.37 ppm) ozone for 2 hours (21). The student nurses studied were healthy young adults relatively free of chronic diseases. These same effects in elderly persons or individuals with chronic lung or heart diseases might produce relatively greater physiologic embarrassment. It is also conceivable that healthy young adults have a greater tolerance to photochemical oxidants. If that is true, the thresholds observed in this study may be even lower for other population groups.

4. Impairment of Performance Associated with Oxidant Pollution

a. Athletic Performance

Wayne et al. (22) have studied the athletic performance in 21 competitive meets of student cross-country track runners at a high school in Los Angeles County, from 1959 to 1964. Oxidant measurements for the hour of the race, and 1, 2, and 3 hours before the race were related to the running time for each athlete. The effects of oxides of nitrogen, temperature, relative humidity, wind velocity, and wind direction were all considered, but did not reveal any relationship to the running times. A significant relationship was observed between oxidant levels and the percent of team members whose performance decreased compared to their performance in the immediately previous home meet, as is shown in Figure 7-5. The correlations between performance and oxidant levels were quite high, but diminished as the interval

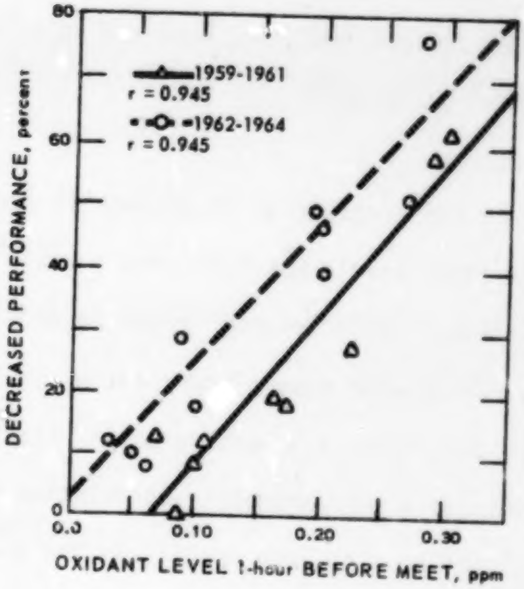


Figure 7-5. Relationship between oxidant level in the hour before an athletic event and percent of team members with decreased performance (22).

between the oxidant measurement and the time of the meet increased. The authors speculated as to possible mechanisms for this association and pointed out that a direct effect on oxygen utilization could occur or that there might be detrimental effects of discomfort from eye and respiratory irritation. The data provide convincing evidence that some component of the air which was measured as oxidant had a causal effect on team performance. With increasing levels of oxidant, there was a manifest impairment of team performance which occurred over a range of oxidant concentrations from 60 to 590 $\mu g/m^3$ (0.03 to 0.30 ppm).

While no threshold level for this effect was specified in the Wayne paper, a subsequent regression analysis by Hasselblad et al. (23), using a "hockey-stick" function, resulted in a threshold estimate of 235 $\mu g/m^3$ (0.12 ppm).

b. Automobile Accidents

A study (24) of the association of automobile accidents with days of elevated oxidant levels was performed to examine the possibility that oxidant pollution may impair performance either directly, by interfering with oxygen transport or utilization, or indirectly, by eye discomfort and respiratory irritation. Ury (24) applied a sign-test and a non-parametric correlation analysis to data for each daylight hour of each weekday in the 3-month period from August through October, for both 1963 and 1965. There was a total of 90 sets of data for testing, covering 9 hours daily and 5 days weekly. The sign-test results of each set were obtained by taking successive pairs of weeks (first week compared with second;

third week compared with fourth, etc.) and scoring a plus (+) if the week with the higher oxidant had more accidents for that set and a minus (-) if it had fewer, and awardin[illegible] tie if the accident frequency or the oxidant levels were eq[illegible]able 7-7). No particular pattern was shown by hours of the day, b[illegible]ttern did appear based on the day of the week.

Table 7-7. SIGN-TEST DATA FOR TESTING THE ASSOCIATION OF OXIDANT LEVELS WITH ACCIDENTS, LOS ANGELES, AUGUST THROUGH OCTOBER, 1963 AND 1965 (24)

Weekday	Year	Plus	Minus	Tie
Monday	1963	17	23	5
	1965	21	21	8
Tuesday	1963	25	14	6
	1965	25	21	6
Wednesday	1963	23	18	2
	1965	24	21	9
Thursday	1963	29	13	8
	1965	27	19	6
Friday	1963	24	25	5
	1965	25	21	6
Total	1963	118	93	26
Total	1965	122	103	35
Total		240	196	61

The data from this study indicate a statistically significant relationship between oxidant levels and automobile accidents. The method of analysis employed in the study does not lend itself to the determination of a threshold level for this effect. Similar results were obtained with the non-parametric correlation test.

These data are consistent with the findings of Lagerwerff (25), in which inhalation of 392 to 980 $\mu g/m^3$ (0.2 to 0.5 ppm) of ozone for 3 hours, or for two 3-hour periods interrupted by a 1- hour rest period, decreased visual acuity in the dark-adaption and middle-vision ranges.

Carbon monoxide, oxides of nitrogen, or particulate matter, which would also have been relatively increased during the periods when oxidant was elevated, could also have contributed to the observed results. Furthermore, increased traffic density at certain times may have caused increases in both oxidant levels and accident rates. Thus, additonal statistical analyses are indicated.

c. Ventilatory Performance and Resistance to Infection

The ventilatory performance, measured by the Wright Peak Flow Meter, of two groups of elementary school children living in the Los Angeles basin was assessed twice monthly for 11 months by McMillan et al. (26). One group of 50 children resided in an area exposed to seasonally high photochemical oxidant concentrations. The other group of 28 children lived in a less polluted area. During the 11 months of the study, no correlations were found between acute changes in photochemical oxidant pollution and ventilatory performance. Contrary to expectation, persistently higher ventilatory performance results were obtained from the children residing in the more polluted of the two communities. Several important differences existed between these two groups of children. The children in which lower ventilatory performance was measured were found to have an incidence of upper respiratory tract illness three times greater that that reported in the children of the less polluted community. A significant ethnic difference was also present: the majority of children in the less polluted community were from a single ethnic group, whereas the children in the other group were ethnically heterogeneous. Thus the expected impairment of photochemical oxidant pollution on ventilatory performance was not found--either because the group differences other than air pollution exposure had a ventilatory performance effect opposite to that of oxidant pollution, or because differences in oxidant exposure of the magnitude found in this study produced no functional impairment. In either case, variations in photochemical oxidant pollution, from daily averages of 100 to 550 $\mu g/m^3$ (0.05 to 0.28 ppm), had no demonstrable effect on the ventilatory performance of the 50 school children living in the more polluted of the two communities.

Cohen et al. (27) examined the effects of oxidant air pollution upon pulmonary function in nonsmoking adults. Seventh Day Adventists were chosen for study since few of them smoke, and they comprised a readily accessible population which was interested in health. The San Gabriel Valley, California, was selected as the high-exposure area, and the San Diego, California, area was chosen as a low-pollution area.

The study population in each area consisted of white English-speaking adults between the ages of 45 and 64 years who had not smoked in the last 20 years and who had a lifetime history of smoking less than one pack per year. Each participant was interviewed by a physician and underwent tests of ventilatory function. Study participants were also requested to complete a respiratory questionnaire which was a modified combination of the British Medical Research Council respiratory questionnaire and the London School of Hygiene cardiovascular questionnaire. Questionnaires were obtained at four sampling cities in each area for 2 months during September 1969 preceding the study, and during January 1970, the month of the study. In addition, records from the Los Angeles County Air Pollution Control District in each area were reviewed to obtain data representative of chronic exposure.

Daily maximal hourly average oxidant levels in the San Gabriel Valley and in San Diego during the time of study were 235 and 137 $\mu g/m^3$ (0.12 ppm and 0.07 ppm), respectively. Measurements of total suspended particulates, respirable particulates and sulfur dioxide were essentially equivalent in both areas during the time of study. A comparison of both study areas during 1963 to 1967 demonstrated that both areas had similar annual mean values for all hourly oxidant concentrations, although the means of the maximal hourly concentrations for San Gabriel (about 274 $\mu g/m^3$ or 0.14 ppm) were approximately twice that found in San Diego (137 $\mu g/m^3$ or 0.07 ppm), and the number of days in which the maximal hourly average exceeded 294 $\mu g/m^3$ (0.15 ppm) was nearly seven times more frequent in San Gabriel than San Diego. Annual averages of other pollutants (carbon monoxide, nitric oxide, nitrogen dioxide, hydrocarbons, sulfur dioxide, and particulates) during 1963 to 1967 were generally higher in San Gabriel than in San Diego.

Results indicated that there were no significant or consistent differences between the two areas in terms of the prevalence of respiratory symptoms. The overall prevalence of chronic bronchitis, defined by the presence of a productive cough on most days for a minimum of 3 months in at least 2 consecutive years, was 1.7 percent. The overall prevalence rate for nonpersistent cough and phlegm (lasting less than 3 months of the year) was 1.2 percent. Similarly, no significant differences were found in measurements of pulmonary function between areas and all measurements were within expected normal values. These measurements included studies of vital capacity and forced expiratory volume. Thus, in this study, significant differences in pulmonary function tests or frequency of chronic respiratory disease symptoms were observed between residents of areas differing twofold in peak values of oxidant air pollution. Possible explanations for this negative finding include the following: 1) exposures may not have been high enough to cause damage; 2) effects might be more closely related to differences in annual means, which were similar in both study areas; 3) effects might be more pronounced among individuals who smoke; and 4) the population studied may have been unusually resistant to the effects of environmental pollution.

The possible effect of chronic exposure to photochemical oxidants upon susceptibility to influenza infection was examined by Pearlman et al. (28) retrospectively during 1968 and 1969 among 3500 elementary school children residing in five Southern California communities. Seven to eight hundred second, fourth, and sixth grade students from upper middle-class socioeconomic areas in each city participated. Selection of these communities was based upon differences in chronic oxidant exposure, although no difference in exposure was present prior to or during the epidemic. Pasadena and Riverside were chosen as the high-oxidant cities, Garden Grove as an intermediate city, and San Diego and Santa Barbara as low-level cities. Seasonal peak mean daily maximum hourly oxidant levels recorded in each city from August to October during 1964 to 1967 were, respectively, 451, 368, 235, 157, and 176 μg/m^3 (0.23, 0.19, 0.12, 0.08, and 0.09 ppm.)

The incidence and duration of influenza-like illnesses between November 1968 and January 1969 was determined by administration of a questionnaire to the parents of these children during March 1969. Parents were questioned as to whether their children had had influenza and, if so, the length of illness and presence or absence of fever, coryza, and myalgia. Occurrence of an influenza-like illness was defined as a febrile case accompanied by coryza and/or myalgia. Blood specimens were obtained by finger-prick from these children during April 1969 for subsequent titration by hemagglutination inhibition (HI) against A_2/Hong Kong influenza and by complement fixation against type A soluble antigen. Complement fixation established a hemoglobin HI titer of greater than or equal to 1:32 as indicative of recent infection with A_2/Hong Kong influenza.

For the entire study, based upon the questionnaire, 72.8 percent of the children reported illness, 35.4 percent being febrile illness and 21.5 percent influenza-like illness. Morbidity within each city did not consistently follow the exposure differences. A high- and a low-exposure city, Pasadena and San Diego, respectively, reported the greatest degree of febrile illness. Pasadena and Santa Barbara, a high- and a low-exposure city, respectively, reported the most influenza-like illness. Adjustment for grade and sex differences by rigid analysis did not reveal any significant differences among cities.

Overall, 24.6 percent of children tested were found to have significant HI titers greater than or equal to 1:32. The percentage of children with significant titers rose from 10.3 percent among those with no illness, to 28.9 percent among those with any illness, to 39.5 percent in those with febrile illness, and to 45.6 percent among those with an influenza-like illness. When the frequency of positive titers among febrile and influenza-like illness categories in the two high-exposure cities were compared with those in the two low-exposure cities, statistically significant city differences inverse to the pollution gradient appeared. More children with illness, that is, symptoms accompanied by elevated titers, were found in the low-pollution cities. Although the two high-pollution cities studied, Pasadena and Riverside, reported higher subclinical illness rates (elevated titers without illness) than the low pollution cities, the differences were not statistically significant.

This study failed to demonstrate any relationship between chronic oxidant air pollution and influenza-like illness. Since animal studies have demonstrated increased morbidity related to acute oxidant exposures, it may be that no effects were found because of a lack of significant acute exposure differences among cities. Perhaps the children in this study did not receive an oxidant dose sufficient to impair host defense mechanisms. Though school children were selected for study, it may be that other high-risk populations, such as those with chronic cardiorespiratory disease, might have demonstrated a pollution-related influenza effect.

Wayne et al. (29) examined the effects of oxidant air pollution upon respiratory illness among children as measured by absentee rates from elementary school. Absentee data were collected from two elementary schools in Los Angeles from September 17, 1962, to June 21, 1963. Reasons for absences were initially obtained from parents and were classified into various categories by teachers. Air pollution measurements were supplied by the Los Angeles Air Pollution Control District. Measurements for school 1, located in east downtown Los Angeles, were made at a station located within 3 km of that school. Air quality data for school 2 were monitored at a station within 3 km of the school during the initial 3 months of study and at a station 6.5 km from the school for the remainder of the study. Oxidant measurements were recorded as maximum hourly averages for each day and as daily averages between 1000 and 1500 hours.

Absence rates in both schools were highest during the winter season, corresponding to the lowest levels of oxidants. No consistent association was observed between weekly mean oxidant levels and weekly absence rates for respiratory disease. Highest absence rates were reported on Mondays and Fridays at both schools; in contrast, oxidant levels were consistently higher during midweek periods. No evidence was obtained for an association between oxidant levels on a given day and absences for respiratory illness on succeeding days, even after standardization to adjust for day-of-week effects upon absence.

In evaluating the negative results of this study, it should be noted that, because differences in oxidant levels at each school were generally within 20 to 40 $\mu g/m^3$ (0.01 to 0.02 ppm), differences in exposure may not have been sufficient to affect rates significantly.

5. Eye Irritation in Relation to Variations in Oxidant Levels

a. Panel Studies

A variety of individuals in various parts of Los Angeles have been studied to determine the occasions and the types of pollutants responsible for eye irritation, one of the most frequently reported symptoms associated with exposure to oxidant pollution (30).

During the first period, observers were asked to report eye irritation on Tuesdays and Fridays. This was later changed to just those days for which eye-irritating levels of pollution were predicted. In general, the observers were office and factory workers; one of the panels consisted of a group of staff members of the California Institute of Technology. The eye-irritation data were compared with instantaneous values of oxidant concentrations as measured by potassium iodide recorders. The data are shown in Figures 7-6 and 7-7 and in Tables 7-8 and 7-9. The "expert" panel (experienced scientists) and the other panel did not significantly differ in the correlations of eye irritation with oxidants, carbon monoxide, particulate matter, and aldehydes. During the second period, August through November of 1955, similar panels observed eye-irritation effects during each day of the work week. In this study, maximum oxidant concentrations were compared with reported eye-irritation effects.

From the data provided by the Air Pollution Foundation studies (30), linear mathematical relationships between maximum oxidant values and mean maximum eye-irritation values were derived. The data from these studies demonstrated increasing eye irritation with increasing concentrations of oxidant pollution over the range of

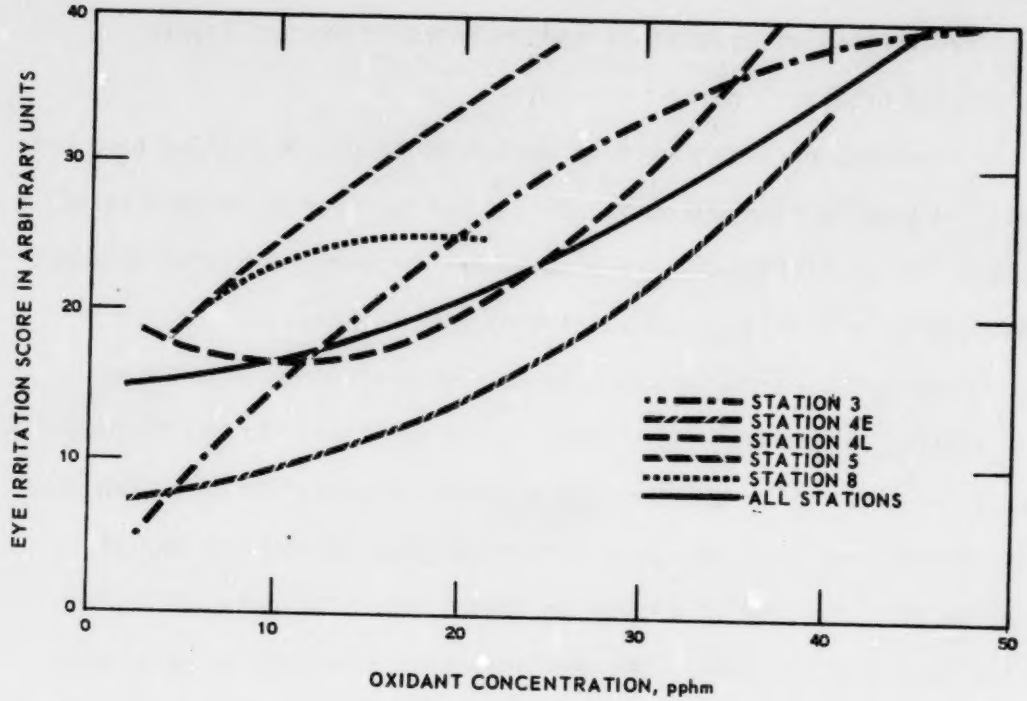


Figure 7-6. Regression curves relating eye irritation and simultaneous oxidant concentrations from a number of stations in the Los Angeles area (30).

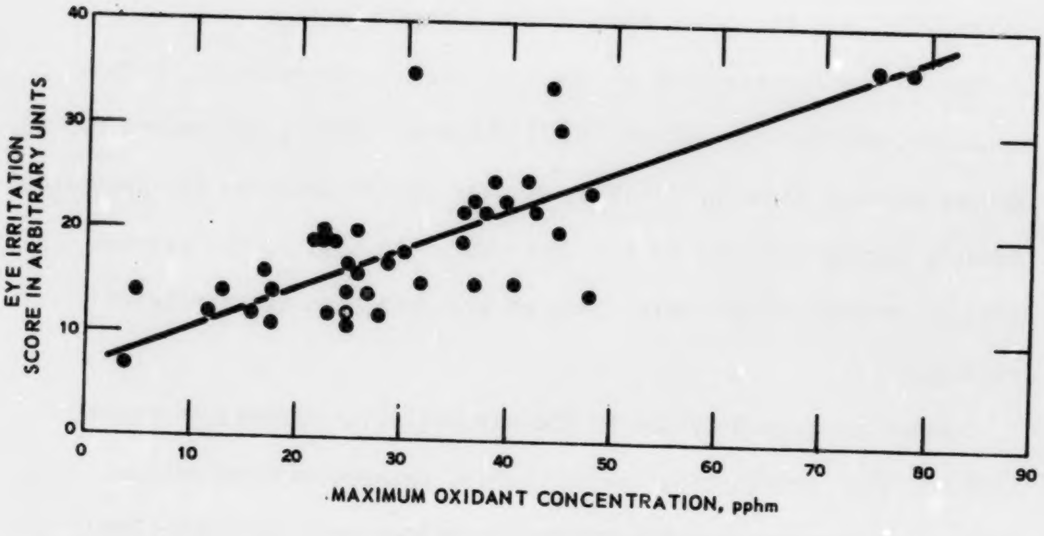


Figure 7-7. Variation of mean maximum eye irritation, as judged by a panel of "experts" with maximum oxidant concentrations, Pasadena, August-November, 1955 (30).

Table 7-8. CORRELATION OF EYE IRRITATION WITH SIMULTANEOUS OXIDANT CONCENTRATIONS IN ORDER OF DECREASING EYE IRRITATION SCORE, FOR A NUMBER OF STATIONS IN THE LOS ANGELES AREA (30)

Station	Number of daily observations	Variance, D (or r^2)	Average eye irritation score	Average oxidant concentration, ppm
5	25	0.88	26.2	0.13
8	29	0.68	22.0	0.10
4L	24	0.76	21.9	0.21
2	30	0.06	21.3	0.11
3	67	0.56	18.2	0.15
4E	66	0.65	13.0	0.17
11	344	0.18	18.8	0.14

Table 7-9. CORRELATION BETWEEN EYE IRRITATION AND SIMULTANEOUS OTHER VARIABLES AS JUDGED BY A PANEL OF "EXPERTS".(30)

Variables	Variance, D or r^2	Average value of variable, ppm unless otherwise noted	Average eye irritation score	Number of observations
Oxidant	0.65	0.17	13.0	66
NO_x	0.07	0.20	13.1	51
CO	0.53	0.27	14.2	47
Hydrocarbons	0.39	0.17	14.0	53
Visibility	0.17	1.2 miles	13.3	56
Particulates	0.53	21.1 Coh units	13.7	26
Aldehydes	0.48	0.19	14.0	38

instantaneous values from 100 to 880 μg/m^3 (0.05 to 0.45 ppm), although no clear threshold level for this effect is apparent (Figure 7-6).

Other studies on eye irritation have been performed, including one in which a panel of employees of the Los Angeles Air Pollution Control District was queried during the period 1955 to 1958 (31). A group of environmental sanitation workers in the San Francisco Bay Area was also studied during the same period. Neither of these panels reported anything other than a tendency to experience increasing occurrence of eye irritation with increasing oxidant levels. As in all such studies, there were some individuals who reported eye irritation even when there was no oxidant present.

b. Student Nurse Study

Hammer et al. (32) reported on respiratory and eye symptoms among two groups of student nurses studied during a 24-day period from October 29 through November 25, 1962, in Santa Barbara and Los Angeles, California. In general, the symptoms were more frequent among the Los Angeles students than among those in Santa Barbara. The choice of period for study was intended to obviate the effects of season or of major respiratory and infectious diseases. The relationships between the mean frequency of symptoms by day as measured by eye irritation, coughing, and sneezing, and the daily maximum oxidant level is shown for the Los Angeles students in Figure 7-8. Unfortunately, a complete set of oxidant measurements was not available. This accounts for the gap in the distribution shown. It is of interest that on one day in which an oxidant level of 450 μg/m^3 (0.23

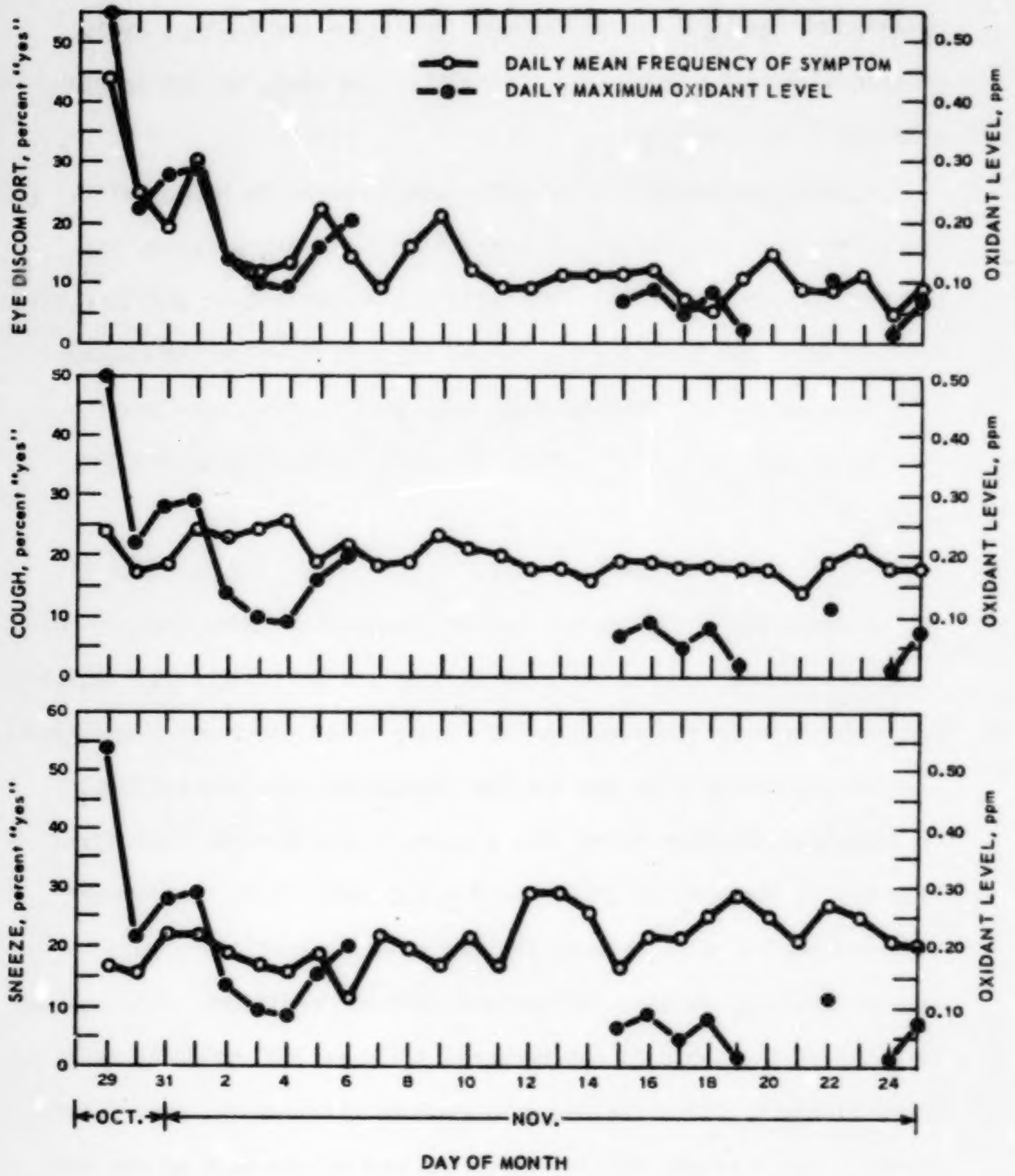


Figure 7-8. Relationship between oxidant concentrations and selected symptoms in Los Angeles, October 29 through November 25, 1962 (32).

ppm) was observed in Santa Barbara, no unusual symptom frequency was reported by the students. Data plotted in Figure 7-8 again show a relationship between increasing eye irritation and maximum daily photochemical oxidant concentrations over the range of 200 to 880 $\mu g/m^3$ (0.10 to 0.55 ppm).

A more comprehensive study of student nurses by Hammer et al. (20) over a greater observation period than in the original study has previously been described. This analysis revealed that the threshold for increased eye discomfort occurred on days in which the maximum hourly oxidant level reached about 300 $\mu g/m^3$. This corresponds to a daily maximum hourly oxidant threshold of approximately 294 $\mu g/m^3$ (0.15 ppm).

c. Evaluation of Filters for Removing Irritants from Polluted Air

A study was conducted to evaluate the sensory effectiveness of air-filter media for removing eye irritants from polluted air (33,34). Eye irritation in two groups of 20 female telephone company employees, similar with respect to age and job characteristics and employed in identical adjacent rooms, was evaluated each workday (123 days) from May to November of 1956. Active and dummy filter units were switched periodically between the two rooms so that the groups were alternately exposed to test and control conditions. The sensory response of the subjects was measured daily at 1100 hours by means of a questionnaire; simultaneous measurements of oxidants, particulate matter, and nitrogen dioxide were obtained within each of the two rooms and immediately outside the building.

The differences in eye irritation betwen the activated-carbon-filtered and non-filtered test situations were in all cases highly significant (Table 7-10). A statistically significant correlation between eye irritation and oxidant concentrations occurred in the non-filtered room (Table 7-11). The scatter diagram of results (see Figure 7-9) suggests an eye irritation threshold as the concentration of oxidants exceeded 200 $\mu g/m^3$ (0.10 ppm).

Table 7-10. EFFECT OF FILTER UPON SENSORY IRRITATION AND CHEMICAL MEASUREMENTS (33)

Test condition	Eye irritation index	Oxidants, pphm[b]	NO_2, pphm
0.032[a] Activated carbon filter			
Mean, non-filtered room	1.99	9.8	1.5
Mean, filtered room	1.01	0.49	0.41
Difference between means	0.98	9.4	1.1
Probability that the difference could have occurred by chance	<0.01	<<0.01	<<0.01
0.016 Activated carbon filter			
Mean, non-filtered room	2.95	8.4	3.4
Mean, filtered room	1.41	1.8	1.6
Difference between means	1.54	6.7	1.8
Probability that the difference could have occurred by chance	<0.01	<<0.01	<<0.01
0.0075 Activated carbon filter			
Mean, non-filtered room	5.45	13.9	2.7
Mean, filtered room	2.35	4.9	5.7
Difference between means	3.10	9.0	3.0
Probability that the difference could have occurred by chance	<0.05	<0.01	[c]
0.0030 Activated carbon filter			
Mean, non-filtered room	2.35	7.3	4.7
Mean, filtered room	1.19	3.6	4.9
Difference between means	1.16	3.7	0.2
Probability that the difference could have occurred by chance	<0.01	<0.01	[c]
Particulate filter			
Mean, non-filtered room	2.13	5.7	6.3
Mean, filtered room	1.91	3.4	5.5
Difference between means	0.22	2.3	0.8
Probability that the difference could have occurred by chance	[c]	<0.01	<0.02

[a] Refers to air detention time in seconds

[b] Measured by the KI method.

[c] Difference not significant.

Table 7-11. PEARSON PRODUCT MOMENT CORRELATION COEFFICIENTS BETWEEN EYE IRRITATION AND ENVIRONMENTAL FACTORS IN A NONFILTERED ROOM (33)

Irritation	vs	Oxidants concentration, by Phenolphtalein method	0.81
Irritation	vs	Oxidants concentration, by KI method	0.81
Irritation	vs	NO_2 concentrations	0.05
Irritation	vs	Particulate	0.15
Irritation	vs	Temperature	0.49
Irritation	vs	Relative Humidity	-0.24
Temperature	vs	Relative Humidity	-0.38
Temperature	vs	Oxidants concentration, by KI method	0.29
Oxidants concentration, by Phenolphtalein method	vs	Oxidants concentration, by KI method	0.88
NO_2 concentrations	vs	Oxidants concentration, by KI method	-0.15

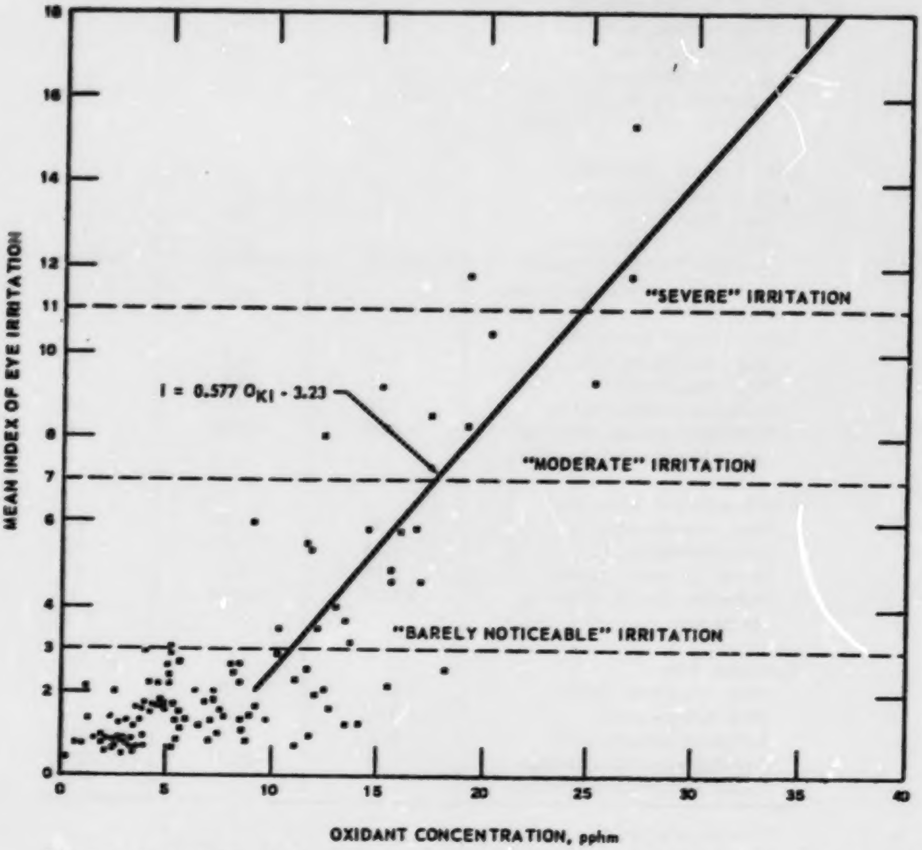


Figure 7-9. Mean index of eye irritation versus oxidant concentration (33).

Nitrogen dioxide concentrations were reduced by the activated-carbon filters during their early use but, after a period of time, nitrogen dioxide concentrations in the filtered atmosphere increased. No significant correlations between eye irritation and nitrogen dioxide levels were observed, nor were significant correlations found between eye irritation and concentrations of particulate matter.

d. Photochemical Oxidant and Eye Irritation in Locations Other Than California

Oxidant measurements at levels likely to be associated with eye irritation have been reported from a number of other cities. Circumstantial evidence of increased eye irritation has been reported in Washington, D.C.; Denver; New York City; and St. Louis. An epidemiologic study of eye irritation was carried out by McCarroll et al. (35) on a population living in mid-town Manhattan. The investigators established a system of weekly health reports by families, based on the presence or absence of certain symptoms. In October 1963, there were substantial increases in the frequency of new reports of eye irritation (increasing from about 2 to nearly 5 percent of the population). Oxidant measurements, made at some distance away, had increased during the period under study. Unfortunately, clear conclusions from these data cannot be drawn. There were high levels of sulfur oxide, particulate matter, and carbon monoxide pollution. It is quite possible that eye irritation symptoms in New York City result from mixed pollution of both the oxidizing and reducing type.

e. Discussion

From these data, it can only be concluded that eye irritation in relation to increased levels of air pollution occurs in other locations in addition to California. It is of great importance that this impression be documented and quantified by additional panel studies.

When interpreting implied relationships associating eye irritation and ambient oxidant levels, care must be exercised in drawing conclusions regarding cause and effect. Experimental studies have shown that ozone, the principal contributor to ambient oxidant levels, is not an eye irritant. Peroxyacyl nitrates have been shown to be powerful eye irritants; even more irritating is peroxybenzoyl nitrate. Formaldehyde and acrolein, also products of the photochemical system, have likewise been shown to produce eye irritation. A postulated explanation for the relationship between ambient oxidant levels and eye irritation is that "oxidant" is a measure of the photochemical activity that produces the aforementioned eye irritants.

C. CHRONIC EFFECTS OF PHOTOCHEMICAL OXIDANTS

To define the chronic effects of prolonged exposure to photochemical oxidant pollution, investigators have contrasted health characteristics of populations living in clean and polluted communities. Results from these studies must be interpreted with caution because the populations of two communities are often different in many respects other than air pollution exposure. In carefully designed epidemiologic studies, the principal determinants of the disease condition being investigated are identified, and population samples from each study area are matched on the basis of those determinants. Thus, information on socioeconomic status, climate, cigarette smoking habits, and occupation must usually be obtained in two-community air pollution studies, and the similarity in these characteristics of the study population must be documented. When differences other than air pollution exposure are present in the study areas, demonstration of the same air pollution-health effect association in studies of other communities is usually required before the association can be firmly accepted.

In general, epidemiologic identification of the chronic effect of air pollution requires replicated studies of large, well-defined populations over relatively long periods of time by competent investigators producing consistent results. These requirements are not adequately met in this section. To prove or disprove the existence of a long-term effect may require an excessively long or hazardous period of exposure and measurement. Studies which suggest that a pollutant may be a health hazard are sufficient to warrant preventive action until such time as the pollutant-health effect association is convincingly proved or disproved.

1. Mortality in Areas of High and Low Oxidant Pollution

a. Lung Cancer Mortality

It is known that active chemical carcinogens are found in polluted atmospheres (36) and that ozone, a principal component of photochemical oxidant pollution, has radiomimetic properties. Beginning in 1957, a prospective study of lung cancer among 69,160 members of a war-veterans group was undertaken in California. This represented between 50 and 60 percent of the membership mailing list of eligible individuals.

This population underrepresents those with chronic illness of long duration, since they may not have been eligible for military service; it probably overrepresents those who are cigarette smokers. The cooperating subjects reported, by postal questionnaire, their residence, occupation, and smoking histories. It thus became possible to carry out a reasonably economical, logitudinal study and to contrast, according to lung cancer experience, short-term with long-term residents of the major metropolitan areas of California. Buell et al. (37) have reported on the first 5 years of follow-up of this population. Identifying data for each individual were maintained on a roster against which the death certificates were checked for the 5-year period 1958 to 1963. Data for mortality from other chronic lung conditions were also available. A total of 336,571 man-years of observation was included in the report.

As is shown in Table 7-12, long-term residents of Los Angeles County have slightly lower age- and smoking-adjusted lung cancer rates than residents of the San Francisco Bay area counties and San Diego County. These urban groups have higher rates than the population residing in the rest of the state. The relative risk of lung cancer for heavy smokers (more than one pack a day) is greater in Los Angeles County than in other areas of California (Table 7-13). For nonsmokers, the rate in the two metropolitan groups is substantially greater than in the rest of the state. The San Francisco-San Diego rates, however, are higher than those in Los Angeles. The duration of exposure necessary to induce lung cancer might possibly exceed the duration of observation from these populations.

Table 7-12. TOTAL LUNG CANCER MORTALITY IN AN AMERICAN LEGION STUDY POPULATION, CALIFORNIA, 1958-1962 (37)

	Los Angeles County		San Francisco Bay Area and San Diego Counties		All other Calif. counties	
	Mortality rate[a]	Total deaths	Mortality rate[a]	Total deaths	Mortality rate[a]	Total deaths
Age-adjusted[b]	95.9	n/a	104.5	n/a	75.3	n/a
Age-smoking adjusted	95.4	n/a	102.0	n/a	75.5	n/a
Resident[c]						
At least 10 yr	96.6	79	106.3	58	79.9	69
Less than 10 yr	76.7	27	69.1	13	68.5	30
Unknown	123.4	12	215.3	10	65.2	6

[a] Deaths per 100,000 man-years.
[b] Age-adjusted by the direct method to the total study population.
[c] Age and smoking adjusted.

Table 7-13. LUNG CANCER DEATHS AND RELATIVE RISKS PER 100,000 MAN-YEARS OF AN AMERICAN LEGION STUDY POPULATION, BY EXTENT OF CIGARETTE SMOKING AND RESIDENCE, CALIFORNIA, 1958-1962 (37)

Cigarette smoking lifetime history[a]	Los Angeles County		S.F. Bay Area and San Diego Counties		All other counties[b]	
	Rate	Relative risk	Rate	Relative risk	Rate	Relative risk
None	28.1	2.5	43.9	3.9	11.2	1.0
Less than one pack	63.6	5.7	77.1	6.9	61.0	5.4
About one pack	126.0	11.3	134.5	12.0	124.9	11.2
More than one pack	241.3	21.5	226.0	20.2	137.5	12.3
Ratio: More than one pack / none	8.6		5.1		12.3	

[a] Age-adjusted by the direct method to the total study population.
[b] Nonsmokers in all other counties taken as unit risk.

Hence, it appears worthwhile to continue the observation.

From these data, it is not possible to demonstrate any effect of oxidant pollution on lung cancer mortality. In view of the radiomimetic properties of ozone, the possibility of a carcinogenic effect by photochemical oxidants must be studied in greater depth.

b. Chronic Respiratory Disease Mortality

Mortality rates for other chronic respiratory diseases reported in the above study (37) were somewhat higher in Los Angeles than in San Francisco and San Diego Counties, particularly among persons resident for 10 or more years in their respective counties (Table 7-14). Studies by Winklestein et al. (38,39), however, have demonstrated the strong effect of socioeconomic level on chronic respiratory disease mortality. For this reason, the reported relationship between chronic respiratory disease mortality and area of residence in California should be considered tenuous until the possible effect of differences in socioeconomic level among these study areas is documented or

Table 7-14. TOTAL CHRONIC RESPIRATORY DISEASE MORTALITY IN AN AMERICAN LEGION STUDY POPULATION, CALIFORNIA, 1958-1962^a (37)

Residency	Los Angeles County		San Francisco Bay Area and San Diego Counties		All other Counties	
	Mortality rate^b	Total deaths	Mortality rate^b	Total deaths	Mortality rate^b	Total deaths
Resident 10 yrs.	38.4	31	28.3	15	45.6	40
Resident less than 10 yrs.	41.2	14	45.6	8	41.3	17
Unknown	139.1	12	59.8	3	39.7	4
TOTAL	46.7	57	34.0	26	44.4	61

[a] Age and smoking adjusted by the direct method to the total study population.
[b] Per 100,000 man-years.

ruled out. Clearly, the data should be substantiated by studies in depth that would consider these and other variables.

c. Discussion

No obvious effect of prolonged exposure to photochemical oxidants on lung cancer mortality rates was evident. A relationship between chronic respiratory disease mortality and photochemical oxidant pollution was suggested in an isolated study in which data on other important variables were not obtained. A considerable amount of further epidemiologic study is needed to test this association.

2. General Morbidity in Areas of High and Low Oxidant Pollution

a. State of California Health Survey

In 1954, weekly studies were initiated to estimate the total morbidity among the general population of California throughout the year (1). In addition, the population aged 65 years and over was studied separately. As shown in Figures 7-10 and 7-11, a significant association of morbidity with periods of high air pollution within Los Angeles County was lacking, even when selected conditions (colds, asthma, hay fever, and respiratory conditions) were considered. The data in Figure 7-10, however, show that the incidence of illness and injury is consistently greater for the population aged 65 years and older in Los Angeles than in the remainder of California. The average weekly incidence was about 7 percent for the non-Los Angeles population and about 10.4 percent for the Los Angeles population, or approximately 50 percent greater. These data are only suggestive of air pollution effects on health, since differences in population density, race, socioeconomic level, and other important factors are known to

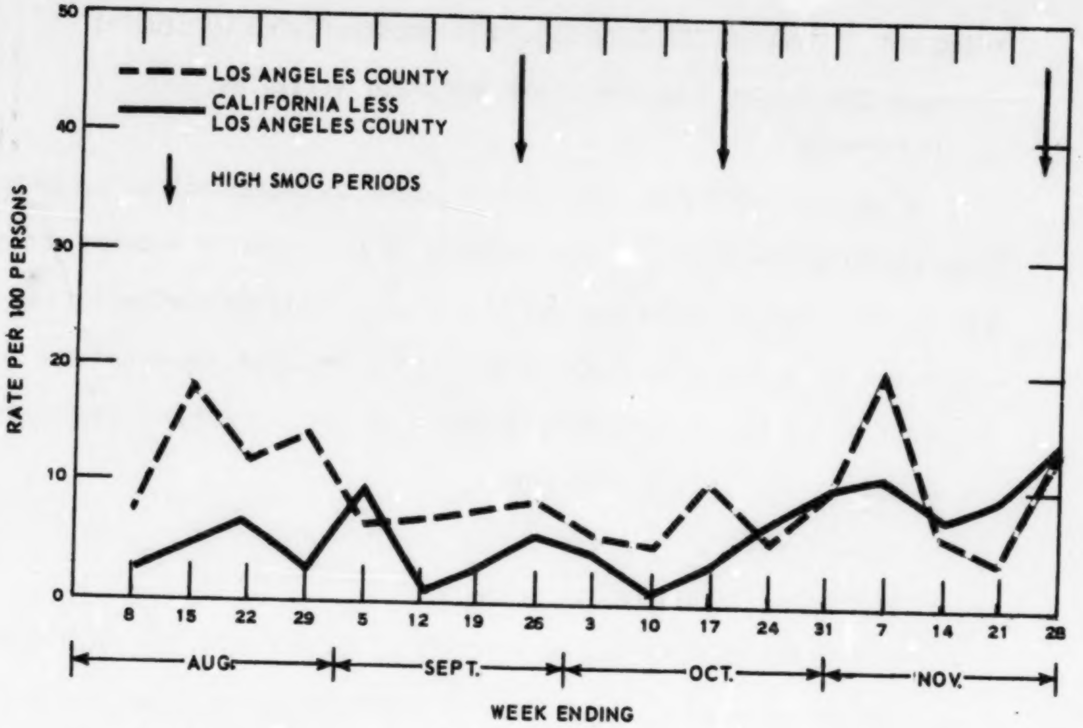


Figure 7-10. Relationship of high smog periods to incidence of illness and injury, persons 65 years and over, August 2-November 28, 1954 (1).

exist between the residents of Los Angeles and those in the remainder of California.

A general health survey was again undertaken in 1956, with one goal being the evaluation of photochemical air pollution in Los Angeles, in the San Francisco Bay Area, and in the rest of the state (40). Because of widespread public interest in air pollution, special precautions were taken to emphasize that this study was undertaken as a general health survey. Special efforts were made to prevent the household interviewers from suggesting the air pollution objective of the study to the subject. The study was designed to explore the following four questions:

1. How extensive is the air pollution problem in Los Angeles in relation to the rest of the state?

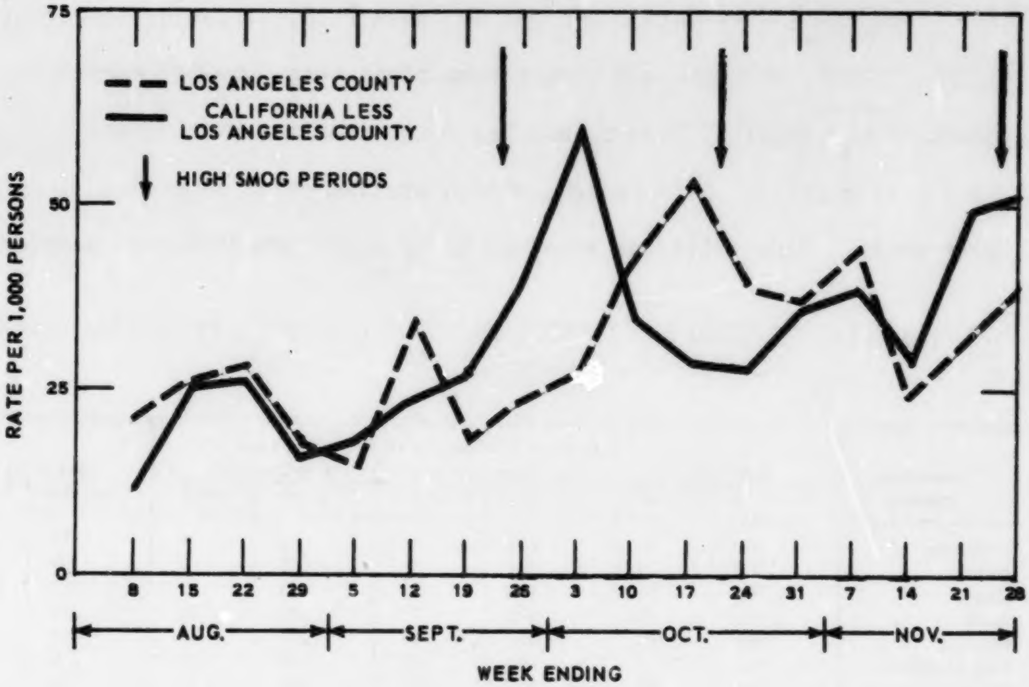


Figure 7-11. Relationship of high smog periods to incidence of selected conditions for persons of all ages, August 2-November 28, 1954 (1).

2. Does air pollution affect a few people much of the time and many people occasionally?
3. Is air pollution a serious source of discomfort?
4. Does air pollution cause dissatisfaction with living conditions in residential communities or in places of employment?

A probability sample of 3545 households was selected as representative of all the people in the state, in the ratio of 1 to 1055. The only populations not included were those in institutions and service camps. Interviewing took place during May and June of 1956. In each household sampled, one adult was randomly chosen and designated for a personal interview. These sub-samples were appropriately weighted.

From the survey data presented in Table 7-15, it can be seen that asthma, cough, and nose and throat complaints were somewhat more frequent in Los Angeles, Orange, and San Diego Counties than in the rest of the state. Differences, though statistically significant, were small. Bronchitis was reported by an equal proportion of sampled persons in the Los Angeles area and in the rest of the state. Sinus conditions and hay fever were most common in the rest of the state. Those persons who admitted to suffering from chronic conditions during the previous year were asked whether they felt worse on some days and, if so, why. The interviewers made no mention of smog or air pollution; the answers that referred to air pollution were considered spontaneous. In Figure 7-12, the responses obtained from residents of Los Angeles County and the San Francisco Bay area are compared. The proportion of the population who attributed a worsening of their condition to air pollution was much greater in

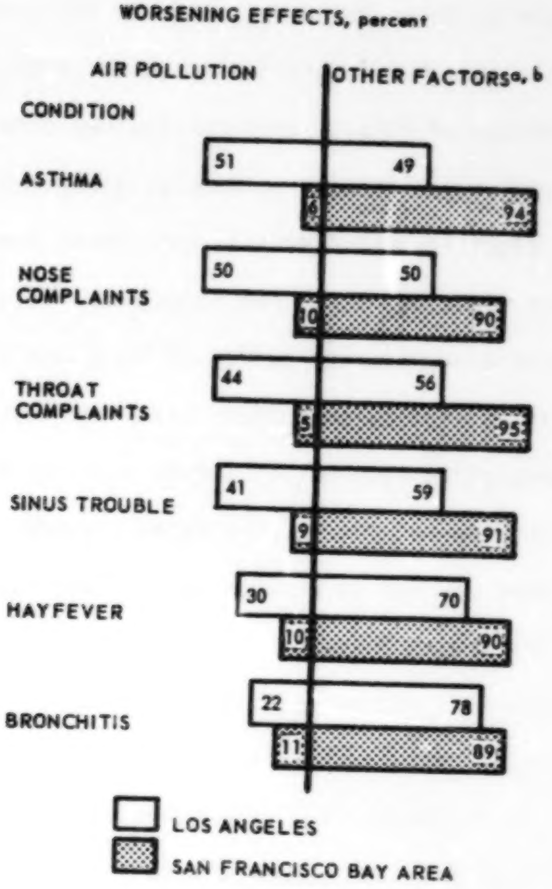
Table 7-15. SELECTED RESPIRATORY CONDITIONS REPORTED BY GENERAL POPULATION SAMPLE, CALIFORNIA, MAY 1956 (40)

Conditions reported	California		Los Angeles, Orange, and San Diego Counties		San Francisco Bay Area Counties[a]		Rest of State	
	Frequency	Percent[b]	Frequency	Percent	Frequency	Percent	Frequency	Percent
Bronchitis	309	4	156	5	71	4	82	5
Asthma	188	3	104	3	45	2	39	2
Cough	1,341	19	746	22	323	17	272	17
Sinus	1,202	17	576	17	302	16	324	20
Hayfever	695	10	265	8	221	12	209	13
Nose complaints	751	11	445	13	186	10	120	7
Throat complaints	848	12	505	15	192	10	151	9
Number of persons interviewed[c]	6,939	100	3,450	100	1,846	100	1,643	100

[a] San Francisco, Alameda, Contra Costa, San Mateo, Santa Clara, Marin, Napa, Solano, and Sonoma Counties.

[b] Percentages will not add to 100 due to reports of multiple conditions or of no conditions.

[c] The general sample of 6,939 persons was drawn from the State's civilian, non-institutional population, age 18 years or older.



[a] Includes the "don't know" responses.

[b] Includes specific foods, overeating, working too hard, not getting enough sleep, emotional upsets, smoking, and presence of other conditions.

Figure 7-12. Air pollution responses for selected conditions obtained from volunteers in Los Angeles and San Francisco Bay areas (41).

Los Angeles than in San Francisco.

The respondents who reported either chronic or repeated attacks of bronchitis, asthma, or cough in the 1956 health survey were selected for a longitudinal study (41). To eliminate youthful allergic asthmatics, the panel was limited to those aged 30 years and older. They were interviewed again on four occasions, twice in 1957, once in 1958, and once in 1959. Data were obtained as to the relative severity and the consequences of illness from respiratory conditions. No patterns of mortality or changes in morbidity were discovered that would indicate significant differences in diverse areas of the state and, hence, that might be attributed to exposure to air pollution.

b. Chronic Respiratory Disease Survey of Telephone Workers

In comparative studies on outdoor telephone workers, Deanne et al. (42) used standardized respiratory survey techniques to study a group of West Coast workmen whose general occupation, medical status, and social status were similar to workmen on the East Coast of the United States and in the United Kingdom. In the older group (aged 50 to 59 years), respiratory symptoms were more frequent in the population of Los Angeles than in that of San Francisco. Persistent cough and phlegm in the 50- to 59-year age group were reported by 31.4 percent of the group in Los Angeles, compared with 16.3 percent in San Francisco. The differences between these two groups could not be accounted for by differences in smoking habits. There were not important differences in the results of pulmonary function tests. In the younger group (aged 40 to 49 years), the frequencies of various respiratory symptoms were not consistently different between Los Angeles and San Francisco residents.

Questions regarding eye irritation were also included. The 49 to 49 age group in San Francisco reported eye irritation about 10 percent of the time; the 50 to 59 age group, about 4 percent. The comparable figures for Los Angeles were 30 percent and 29 percent, respectively. Perhaps more important is the fact that over 50 percent of those in both age groups in San Francisco had never experienced eye irritation, whereas the corresponding figure for Los Angeles was less than 10 percent.

c. Discussion

Several surveys suggest a higher incidence of both chronic respiratory disease symptoms and of other symptoms, including asthma and nose and throat complaints, among residents of Los Angeles than in other areas of California, including the San Francisco Bay area.

3. Effects of Photochemical Oxidant Pollution on Community Satisfaction

a. State of California General Health Survey

At the end of the general health survey undertaken in California in 1956 and previously described (40), direct questions concerning the effects of air pollution were asked. Seventy-five percent of the surveyed population from Los Angeles County was "bothered" by air pollution, in contrast to 24 percent in the San Francisco Bay area, and 22 percent in the rest of the state. These contrasts were also reported for the working populations from these areas (Table 7-16). Of the total number who were bothered by air-pollution, 17 percent in Los Angeles considered moving because of it, in contrast to 4 percent in San Francisco. Of the same total number, 9 percent in Los Angeles considered changing jobs because of air pollution, in contrast to 3 percent in San Francisco (Table 7-17). About 20 percent of the state's residents who had moved out of an air polluted area said the pollution had some influence on their decision to move; 4 percent gave air pollution as their sole reason for moving. Among

Table 7-16. PERCENT OF SURVEY RESPONSES OF GENERAL AND WORKING POPULATIONS "BOTHERED" BY AIR POLLUTION, BY MAJOR GEOGRAPHIC AREAS IN CALIFORNIA, MAY 1956 (40)

Responses	California	Los Angeles County	San Francisco Bay area	Rest of State
General population sample	6,939	2,892	1,846	2,210
	Percent[a]			
Not bothered by air pollution	55	24	76	78
Bothered by air pollution				
Either at home or work	45	75	24	22
Both at home and at work	14	27	4	4
At home only	24	39	14	13
At work only	7	8	6	5
Total at home	38	66	18	17
Total at work	21	35	10	9
Working Population Sample	3,732	1,577	1,028	1,127
	Percent[a]			
Not bothered by air pollution	51	20	71	73
Bothered by air pollution				
Either at home or work	49	80	29	27
Both at home and at work	25	49	7	9
At home only	12	16	11	8
At work only	12	15	11	10
Total at home	37	66	18	17
Total at work	38	65	18	18

[a] Percents are rounded independently.

those who moved from California communities because of air pollution, 75 percent had moved out of Los Angeles County, 8 percent had moved out of the San Francisco Bay area, and 17 percent had moved out of other areas of the state. The reasons given for all of these moves are listed in Table 7-18. Air pollution was given as the reason for 13 percent of the moves from Los Angeles County since 1947. For other areas of the state, the proportion of moves attributed to air pollution was negligible.

Eye irritation appeared to be the most frequently reported effect of air pollution; in some instances, this symptom was accompanied by

Table 7-17. PERCENT OF SURVEY RESPONSES OF GENERAL AND WORKING POPULATIONS, "BOTHERED" BY AIR POLLUTION, WHO HAVE CONSIDERED MOVING OR CHANGING THEIR JOBS FOR THIS REASON, BY MAJOR GEOGRAPHIC AREAS IN CALIFORNIA, MAY 1956 (40)

Responses	California	Los Angeles County	San Francisco Bay Area	Rest of State
General population sample, bothered by air pollution	2,616	1,904	326	386
	Persons bothered, percent			
Have given serious consideration to moving	15	17	4	12
Have not given serious consideration to moving	85	83	96	88
Total	100	100	100	100
Working population, sample workers bothered by air pollution at work	1,410	1,012	190	208
	Persons bothered, percent			
Have given serious consideration to changing jobs	8	9	3	3
Have not given serious consideration to changing jobs	92	91	97	97
Total	100	100	100	100

Table 7-18. REASONS FOR MOVING FROM THREE AREAS OF CALIFORNIA IN RESPONSE TO GENERAL POPULATION SURVEY, MAY 1956 (40)

Reason for moving from area	Los Angeles County, percent		San Francisco Bay Area, percent		Rest of State, percent	
	Prior to 1947	1947-56	Prior to 1947	1947-56	Prior to 1947	1947-56
Financial	62	51	52	51	64	60
Personal	32	27	32	39	27	28
Health	4	3	6	2	2	5
Air Pollution	2	13	-	-	-	1
Weather	-	6	10	8	7	6
Total percent	100	100	100	100	100	100

nasal irritation (Table 7-19). About 20 percent of the respondents expressed a dissatisfaction with the communities in which they lived. Air pollution had not been mentioned at this point in the interview. The reasons for community dissatisfaction are shown in Table 7-20. A far greater proportion of dissatisfied Los Angeles residents (32 percent) attributed their dissatisfaction to air pollution than did residents of the San Francisco Bay area (1 percent) or those in the rest of the state (6 percent).

b. Survey of Los Angeles Physicians

A joint committee carried out a survey of Los Angeles physicians in December 1960 (43). A sample representing 1 in 16 of those physicians registered to practice in the county during 1958 was drawn,

Table 7-19. AIR POLLUTION EFFECTS
REPORTED IN GENERAL POPULATION SURVEY, BY TYPE OF COMMUNITY AND
BY MAJOR GEOGRAPHIC AREAS IN CALIFORNIA, MAY 1956 (40)

	California		Los Angeles County		San Francisco Bay Area		Rest of State	
Air pollution effects	At home	At work	At home	At work	At home	At work	At home	At work
General population sample	6,939	3,732	2,892	1,577	1,846	1,028	2,201	1,127
Respondents bothered by air pollution	2,616	1,410	1,904	1,012	326	190	386	208
Percent bothered by air pollution	38	37	66	64	18	18	17	19
	Persons bothered, percent							
Air pollution effects cited:								
Eyes, effects	75	76	89	88	38	39	41	51
Eye irritation	44	46	54	53	17	18	23	30
Eye and nasal irritation	23	24	26	27	15	14	14	19
Eye irritation and annoying	5	3	6	4	4	3	3	2
Eye, nasal irritation, and annoying	3	3	3	4	2	4	1	-
Nasal irritation, eye not mentioned	10	9	5	4	22	23	22	21
Nasal irritation	8	8	4	3	19	19	18	21
Nasal irritation and annoying	2	1	1	1	3	4	4	-
Annoying only	5	7	2	3	17	26	10	8
Other effects, only	5	2	2	1	5	5	17	4
No effects reported	5	6	2	4	18	7	10	16
Total percent	100	100	100	100	100	100	100	100

Table 7-20. EFFECTS OF AIR POLLUTION ON COMMUNITY SATISFACTION, REPORTED IN GENERAL POPULATION SURVEY, BY MAJOR GEOGRAPHIC AREA, CALIFORNIA, MAY 1956 (37)

Reason volunteered for dissatisfaction with community	California	Los Angeles County	San Francisco Bay area	Rest of state
Dissatisfied, total number	1,345	612	327	406
Climatic	30	41	18	27
Air pollution	16	32	1	6
Weather	14	9	17	21
Nonclimatic	64	54	74	69
Miscellaneous	6	5	8	4
Total percent	100	100	100	100

resulting in a sample of 526 from a total of 9228 physicians. A pretested questionnaire was mailed with a letter signed by the chairman of the air pollution subcommittee. A follow-up was also mailed, and telephone calls were made to the offices of those physicians who had not responded.

Three-hundred fifty of the questionnaires were returned. Of those, 307 (58 percent) were completed and tabulated. The words "air pollution" did not appear in the questionnaire, although bias could have been introduced by the fact that the chairman of the air pollution subcommittee attached a letter to the questionnaire. Seventy-seven percent of the physicians believed that air pollution adversely affected the health of their patients. Two-thirds of the responding physicians felt that air pollution was a factor adversely affecting chronic respiratory disease. One-third of the physicians had advised one or more of their patients to leave the Los Angeles area for health reasons; air pollution was a factor mentioned in two-thirds of these instances. By extrapolation from the sample, assuming it to be representative, it was estimated that physicians had advised over 10,000 patients to move; it was reported that approximately 25 percent of the patients had done so. Nearly one-third of the physicians had themselves considered moving from the Los Angeles area because of air pollution. Among other environmental factors mentioned were overcrowding and traffic congestion, but these were of very small magnitude in relation to the reported difficulties with air pollution.

D. SUMMARY

Epidemiologic studies have been conducted to determine the relationship of photochemical air pollution with mortality, hospital admissions, aggravation of respiratory diseases, impairment of human performance, irritation of the respiratory tract, and eye irritation. The effects of prolonged oxidant exposure on mortality, morbidity, ventilatory function, and community satisfaction have also been studied.

1. Review of Results from Cited Studies

No convincing relationship was observed between short-term variations in photochemical oxidants and daily mortality or hospital admissions, although there was a suggestion that mortality could be related to oxidant levels.

A study of 137 patients with asthma demonstrated significantly more asthma attacks on days when photochemical oxidant maximal hourly concentrations exceeded 430 $\mu g/m^3$ (0.25 ppm).

Chronic respiratory disease patients who were removed from an ambient atmosphere of elevated oxidant concentrations to a room from which pollutants were filtered have shown improvement in ventilatory function. In two other studies, no significant association was found between variations in ambient oxidant levels and changes in respiratory symptoms or function in patients with chronic respiratory disease.

The team performance of high school cross-country track runners was impaired on days of elevated oxidant concentrations measured 1 hour before the commencement of each race; hourly oxidant concentrations ranged from 60 to 590 $\mu g/m^3$ (0.03 ppm). The threshold for this effect has subsequently been estimated at about 235 $\mu g/m^3$ (0.12 ppm). These findings may possibly be related to a pulmonary irritation effect similar to that which has been shown to occur by Bates et al. (44) at higher levels of exposure but at lower levels of exertion. Significantly more automobile accidents have also occurred on days of high oxidant concentrations.

A study of student nurses in Los Angeles has demonstrated a threshold for increased cough and chest discomfort at daily maximum hourly-average oxidant concentrations between 500 and 600 $\mu g/m^3$ (0.25 and 0.30 ppm). This same study also observed a threshold for increased eye discomfort on days with maximum hourly-average oxidant concentrations at about 300 $\mu g/m^3$ (0.15 ppm). These results are consistent with those from other studies which have shown that eye irritation appears to increase when oxidant concentrations exceed 200 $\mu g/m^3$ (0.10 ppm).

Eye irritation, at times accompanied by nasal irritation, was the most frequently reported nuisance effect of air pollution in California. A postulated explanation for the relationship between ambient oxidant levels and eye irritation is that the level of oxidants is a measure of the photochemical activity which produced the eye irritants.

Lung cancer mortality rates were similar among California residents studied in both high- and low-oxidant-pollution areas. A relationship between noncancerous chronic respiratory disease mortality and long-term photochemical oxidant exposure has been suggested in an isolated study in which other important variables were not analyzed. Factors other than oxidant exposure could well have accounted for these observations, and considerable documentation from other epidemiologic studies is required to substantiate these findings. Several surveys have also reported a higher incidence of both chronic respiratory disease symptoms and other respiratory symptoms, including asthma and nose and throat complaints, among residents of Los Angeles than in residents of other areas of California.

A significantly larger proportion of Los Angeles residents has been subjectively bothered by air pollution than of residents of the San Francisco Bay area and the rest of the state. A larger proportion of residents who were bothered by air pollution have considered moving or have moved from Los Angeles than have residents of other areas of California. One-third of the physicians sampled in the Los Angeles area had advised one or more of their patients to leave the area for health reasons, and nearly one-third of the physicians had themselves considered moving from Los Angeles because of air pollution.

2. Future Research Needs

Relatively scanty information has been gathered on community health effects of photochemical oxidants. The relatively localized nature of the oxidant problem accounts for this gap in knowledge. Data reported in this chapter need to be augmented, particularly with regard to lung cancer mortality and the prevalence of chronic respiratory disease. The known irritant potential of ozone, a major component of photochemical pollution, should be explored to establish more exactly how exacerbations of illness in asthmatics and chronic bronchitics on days of elevated oxidant concentrations may be related to the presence of ozone. Interactions of ozone with other pollutants such as SO_2 also require further study. Studies of the acute effects of oxidant pollution could be further refined by careful selection of communities in which differences in exposure to photochemical oxidant levels are maximal. Both chronic and acute effects associated with air pollution exposure can be identified more readily through such studies.

Systemic effects of oxidant exposure were suggested in the athletic performance study, in the study on accident frequency, and in the study on the effects of ozone upon visual acuity. Replication and extension of these studies are clearly indicated. Physiologic and psychomotor tests to elucidate pathological mechanisms for these effects would add important confirmatory evidence and point the way to the determination of valid dose-response relationships.

Well-designed prevalence studies in clean and polluted communities would provide useful quantitative data on the prevalence of chronic respiratory disease, heart disease, and possibly chronic eye pathology in relation to prolonged oxidant exposure. The effect of oxidant exposure on growth and development in utero and in the first years of life is clearly a feasible prospective study. Such a study, suggested by existing toxicology data, might provide important information on the action of this pollutant during a period of life when man is highly sensitive to environmental influences.

Further evaluation of the radiomimetic character of ozone is also indicated, particularly with respect to the possible extrapulmonary and genetic implications of this property.

The impairment by oxidant pollution of immune mechanisms of response to infectious agents, suggested in animal studies, has not been explored in community studies. Classical epidemiologic and laboratory methods for studying acute respiratory illness rates in populations could be applied profitably to the photochemical oxidant problem.

3. Discussion

Accumulated studies reviewed in this chapter reveal that epidemiologic information on the health effects of photochemical oxidant pollution is inadequate. Consistent results for some effects, obtained by various investigators under varying conditions of exposure, are lacking. The few demonstrated associations between oxidant exposure and health effects, such as asthma, pulmonary function, or athletic performance, are inadequate to establish with confidence minimum threshold levels for each effect.

Reported studies suggest, however, that photochemical oxidants are potentially hazardous environmental contaminants. Subjects with chronic respiratory disease seem to be those most threatened by such exposure, but virtually all segments of the population may experience eye irritation at levels of oxidants frequently present in the ambient air. Hazards to normal respiratory function, optimum athletic performance, and safe automobile driving have also been suggested. An association, although not necessarily a cause-effect relationship, has been shown to exist between ambient levels of photochemical oxidants and eye irritation. Since one of the objectives of air pollution control is to promote good health and minimize exposures to potentially hazardous pollutants, the information provided by reported studies cannot be discounted.

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APPENDIX

Conversion Factors
(25°C, 760 torr)

Hydrocarbons expressed as CH_4

$$\text{ppm} \times 655 = \mu g/m^3$$
$$\mu g/m^3 \times 0.00153 = \text{ppm}$$

Ozone or oxidant expressed as O_3

$$\text{ppm} \times 1960 = \mu g/m^3$$
$$\mu g/m^3 \times 0.000510 = \text{ppm}$$

Peroxyacetylnitrate (PAN)

$$\text{ppm} \times 4950 = \mu g/m^3$$
$$\mu g/m^3 \times 0.000202 = \text{ppm}$$

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